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ACUTE TRAUMATIC DISLOCATION OF THE TENDON OF THE LONG HEAD OF THE BICEPS BRACHII

A REPORT OF SIX CASES WITH OPERATIVE FINDINGS

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TRAUMATIC dislocation of the tendon of the long head of the biceps brachii has always been regarded as a lesion of infrequent occurrence. Codman, for example, in his excellent book, *The Shoulder*, lists it under the rare lesions of the shoulder joint.

Our interest in this condition was awakened in 1932 by the first case herein reported and in which our diagnosis was later confirmed by operation. Shortly thereafter, three additional cases were observed and these four cases were embodied in a report made by us at the meeting of the American Orthopaedic Association in June, 1934.* At that time we were unable to discover any case recorded in medical literature in which the diagnosis was afterwards proved by operation. In discussing this report, Gallie and Speed both stated that they had operated upon similar cases. More recently we have had two additional cases which we are including in this report. From this experience we believe that the lesion is not as rare as was formerly supposed and is a condition which should always be borne in mind in the diagnosis of disabilities affecting the shoulder joint.

LITERATURE

The first reported case of dislocation of the tendon of the long head of the biceps brachii muscle appears to have been in a work published in 1694 by William Cowper, entitled *Myotomia Reformata*. He related

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the case of a woman who, while wringing clothes, felt something displace itself at the shoulder. On examination three days afterwards, he noticed a depression of the external part of the deltoid, accompanied by rigidity in the lower part of the biceps and inability to extend the forearm. The following day he manipulated the arm in various directions, whereupon the supposedly dislocated tendon slipped into place and the patient at once recovered the use of the arm. In the following century Cowper's case reappeared in the literature, both with and without recognition of its origin, and the possibility of this lesion was accepted by such influential teachers as Boerhaave and Bromfield. But Cowper's observation became subject to suspicion in the minds of several authors in the next two centuries because of the notoriety which he gained by the publication of his later work on anatomy. The plates of this famous elephant folio were appropriated from Godfried Bidloo, the Dutch anatomist, a not uncommon practice in those days, but the plagiarism was judged of little moment by Boerhaave on account of the slender value of Bidloo's text.

A century later, in 1803, Monteggia reported a second case resembling somewhat in its clinical features that of Cowper except for the dislocation's being habitual, and from that time on until 1910 numerous additional clinical records and articles appeared at intervals. Some of these were highly disputatious; among them may be mentioned those by Jarjavey, Soden, Green, Gerster, White, Marsh, Robinson, Haberern, and Parkhill.

The subject became one of considerable controversy, many frankly denying the possibility of such a lesion. The role played by the long head of the biceps in the so-called partial dislocation of the humerus (Astley Cooper), the effect of arthritis (Adams), and the question of a subacromial bursitis (Jarjavey) figure prominently in the criticisms offered by Bardeleben, Volkmann, Pouteau, von Pitha, Malgaigne, and Chassignac. Soden's case, reported in 1841, is important and unique in that it is the only clinical case which was proved by necropsy, although several examples had been described in which a dislocation was found in dissection material, notably by Stanley.

In 1884, White published a comprehensive review of the literature and, after concluding that cases previously reported "fail altogether to carry conviction," included a clinical report of some length on a case which he had personally observed. The obvious errors and misconceptions presented by White in this analysis have been ably pointed out by Meyer.

Since the time of White's report, the subject of dislocation has been kept alive and renewed mainly by the various anatomical and surgical texts, rather than by practical experience. The recent text by Homans contains no more than the statements: "Several important tendons which run in bony grooves are occasionally dislocated by violent and un-

usual movements. The long tendon of the biceps may be dislocated outward or inward by movements which combine forcible flexion of the forearm with rotation at the shoulder as in wringing clothes. The displacement prevents extension of the forearm until the tendon is replaced by abduction or adduction as the case may be. The tendency to displacement may become habitual.'' The inaccuracies of this statement are obvious and we seem to hear Cowper's case echoing down through 250 years—the wringing of clothes.

More recently in 1926 and 1928 valuable contributions to our knowledge of this subject were published by Meyer. In his later publication Meyer discusses his observations based on a total of thirty-nine instances of spontaneous dislocation of the long tendon of the biceps brachii and on twenty examples of total destruction of the capsular portion of this tendon. These studies carried out on dissecting-room material were a continuation of a series published in the years 1913, 1915, 1921, 1922, and 1924 on destruction of tendons and joints ''from use.''

In only six of the thirty-nine cases was the dislocation maximal; that is, the long head had passed completely over the lesser tuberosity, the remaining cases representing transitional stages. The subjects were of both sexes, the majority Caucasian, and all presumably belonged to the laboring classes. The ages ranged from 50 to 80 years and because of the nature of the material clinical histories were totally lacking.

Meyer concludes that attrition, particularly following the use of the extremity in abduction and lateral rotation, leads to a gradual stretching, detachment, or destruction of the capsule proximal to and in the region of the lesser tuberosity. As a consequence of the weakening of the capsule, which he considered to be the chief restraint to displacement, dislocation ensued. He employs the term ''spontaneous'' to convey the idea that external trauma, accident, or disease is not necessarily involved in these cases. In an earlier paper he states in this connection: ''It is doubtful whether severe or sudden strain, even if prolonged or repeated, can produce the dislocation. . . . Probably strenuous and long continued use resulting in capsular defects alone can produce the condition necessary for its occurrence.''

APPLIED ANATOMY

The general position of the long head of the biceps, from its origin at the supraglenoid tubercle and labrum glenoidale to its course through the intertubercular (bicipital) sulcus, is so well known as scarcely to warrant description here (Fig. 1). There are, however, some details of anatomy and of function which it seems desirable to discuss.

Traditional teaching has it that the tendon is held in place within the sulcus by means of the transverse humeral ligament, a statement which is somewhat misleading. The relative unimportance of the transverse humeral ligament is fully recognized by Meyer, who says that he has

found "this structure either too weak or entirely absent and that transverse fibers bridging the sulcus, which could be properly described as a ligament, seldom are present, and when this is the case, they are found lower down than the so-called ligament of Brodie is supposed to occur." In our opinion the chief structure retaining the tendon within that part of the sulcus which lies below the level of the summits of the greater

Fig. 1.—The tendon of the long head of the biceps brachii in relation to the bicipital groove.

and lesser tubercles is a tendinous expansion from the insertion of the sternocostal portion of the pectoralis major muscle. This expansion, which forms a falciform margin at the deep aspect of the main tendon, is attached to both lips of the sulcus and blends above with the capsule of the shoulder joint. As a rule a few, relatively weak, transverse fibers strengthen the roof so formed. We have often observed, at operation, that the retinaculum so formed is, however, somewhat more substantial

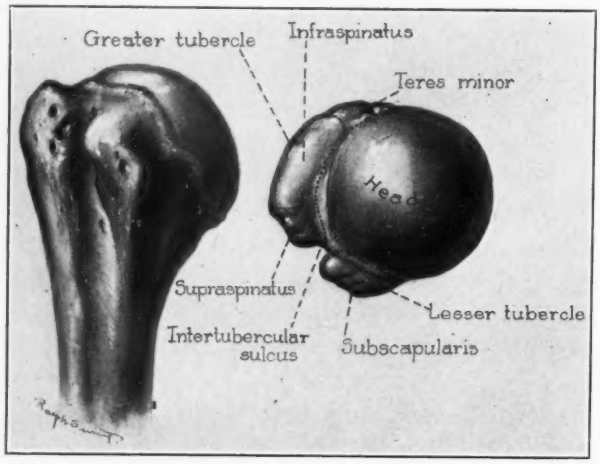
than Meyer's comments would lead one to believe. The most important feature in retention of the tendon is that portion of the capsule of the shoulder joint which bridges the tubercles in the uppermost part of the sulcus. In this region, the capsule is thick and strong and is reinforced by the blending of the neighboring tendons and fasciae. This portion of the capsule is the first and chief obstacle to forward dislocation of the tendon and Meyer found that in all of his cases it had been torn or stretched. Codman, in commenting on Meyer's work, states that, in his opinion, displacement of the tendon is the result of rupture of that portion of the musculotendinous cuff which is inserted into the inner side of the intertubercular notch. This portion is made up of the joint edges of the supraspinatus and subscapularis tendons. (Figs. 2 and 3.)

Occasionally other anatomical features in the construction of the sulcus may exist, such as the presence of or absence of a supratubercular ridge and the varying depth of the sulcus. The supratubercular ridge is described by Meyer as a ridge which extends forward and downward from the region of the articular cartilage to the upper and dorsal portions of the lesser tuberosity. Quoting Cilley, he states that it occurred in 17.5 per cent of 200 humeri. This ridge, when present, must play an important role in dislocation as its distal extremity is on a level with the lesser tubercle and therefore it mechanically diminishes the effectiveness of the tubercle as a trochlea. The depth of the sulcus is an extremely variable factor, depending upon the degree of development of the tubercle and the lips of the sulcus (Fig. 2).

The anatomical factors which favor dislocation of the tendon are well summarized by Meyer: "In all normal joints there are three, and there may be six factors which favor dislocation of this tendon: (1) the normal course of the intracapsular portion of the tendon and its relation to the humeral head, (2) the much greater width of the proximal portion of the tendon, (3) the fact that the anterior wall of the sulcus which is formed by the lesser tuberosity normally acts as a trochlea for the tendon in the usual position of medial rotation, (4) a supratubercular ridge may be present, (5) the capsular attachment may be weakened by intracapsular bursae, and (6) the capsular attachment to the anatomic neck in the region proximal to the lesser tuberosity may be restricted."

The relation of the tendon to its groove varies considerably with different positions of the arm. In medial rotation the tendon is applied to the lateral wall of the sulcus and the adjacent capsule. In slight lateral rotation it occupies the floor of the sulcus. In these positions, therefore, there is no tendency for dislocation to occur. As lateral rotation is increased, the tendon is forced against the medial wall of the sulcus, especially that part formed by the lesser tubercle and the capsular attachment proximal to it. It is of some importance in the mechanism of dislocation to note the changes in angulation of the tendon with varying degrees of abduction or flexion. With the arm at the side, the angula-

tion of the tendon, as it lies in the uppermost part of the groove, approaches a right angle. As abduction is increased, the angulation progressively disappears until at and above 90 degrees of abduction the tendon pursues an almost straight path from its origin at the supraglenoid tubercle to its muscular belly. In consequence, as may be ob-



A. *B.*

Fig. 2.—*A*, Contour of the bicipital groove in relation to the greater and lesser tuberosities of the humerus. In this specimen the upper part of the groove is unusually deep. *B*, Relation of the bicipital groove to the insertion of the tendons of the supraspinatus and the subscapularis.

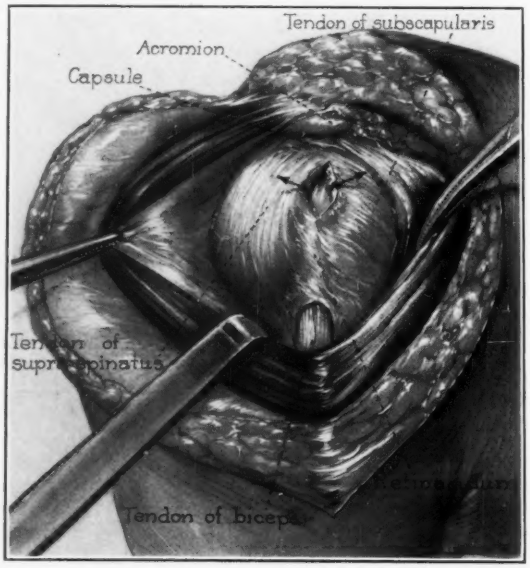


Fig. 3.—Shows the strong, musculocapsular retinaculum bridging the bicipital groove.

served upon examination, there is in the higher ranges of abduction or flexion little tendency for the tendon to slip from its groove. In these positions the tendon, being more nearly straight, parallels the axis of rotation. On the other hand, as the arm approaches the side and the

angulation increases, the tendon is thrown more and more against the lesser tubercle, which then acts as a trochlea. It is in these, the lesser ranges of movement, that the tendon depends for its security in greater measure on the lesser tubercle and the fibrous, anterior roof of the sulcus. At 45 degrees or less of abduction or flexion, the tendon can be most readily displaced from its groove by lateral rotation.

FUNCTION OF THE LONG HEAD OF THE BICEPS BRACHII MUSCLE

Analysis of the action of the component muscles participating in the production of motion at the shoulder joint is notoriously difficult and there is great conflict of opinion, notably in regard to details of action of the individual muscles. There can be no question that much of this conflict arises from failure to appreciate the correct sequence of motion as it occurs in elevation of this extremity.

The common teaching has it that, when the arm is raised, movement up to the right angle occurs at the scapulohumeral joint and that, thereafter, further elevation of the limb is brought about by rotation of the scapula. This teaching, patently incorrect, is nevertheless of wide acceptance both by anatomists and clinicians. Any analysis of the action of muscles about the shoulder based upon the assumption that the above teaching is correct or upon the assumption that the scapula is fixed, as in the recently published experiments of Howard and Eloesser, is bound to lead to erroneous inferences in many important details. For example, we find Howard and Eloesser reaching the remarkable conclusion that the subscapularis muscle acts as an abductor beyond 45 degrees of abduction. Such a finding is not surprising under the conditions of their experiments. When the scapula is fixed, as in their "phantom" model, and the humerus abducted beyond 45 degrees, the insertion of the subscapularis muscle is carried above the axis of movement and the muscle necessarily becomes an abductor. In the living subject, however, it is predominantly scapular rotation which carries the arm to the right angle and true scapulohumeral rotation occurs, for the most part, only above this level. For this reason, it is extremely doubtful that the moment of action of the subscapularis muscle ever rises above the fulcrum at the shoulder joint, even when the arm is carried to 180 degrees, and therefore dubious that, under functional conditions, this muscle ever acts as an abductor of the arm. Furthermore, as pointed out by Lockhart, it is impossible to abduct the arm of the living subject beyond 15 degrees if the scapula has been fixed. For such reasons we find to be unacceptable any opinions on muscle action at the shoulder which neglect the normal sequence of movement. Therefore, an essential preliminary to a discussion of the function of the long head of the biceps is the mention of some of the features which characterize motion at this joint.

The shoulder joint itself, that is the scapulohumeral joint proper, when in motion simply plays a part in a complex arrangement of sev-

eral joints. Careful inspection and palpation of the living subject, especially when combined with fluoroscopic examination, reveal some of the details of the sequence of motion which occur in raising the arm from the side. These methods show that, in motion, the clavicular, scapular, and humeral joints all participate from the very commencement to the termination of the act in a continuous and harmonious movement.

Briefly, the sequence of motion is that at the very inception of the act of raising the arm, the scapula commences to move outward. By the time the arm is at right angles to the trunk, scapular rotation is almost complete, while at the same time but little motion has occurred at the shoulder joint. Our measurements demonstrate that approximately only one-sixth of the available range at the shoulder joint occurs in movements up to the right angle. In carrying the arm above the head to the perpendicular, motion occurs mainly at the shoulder joint, accompanied, however, by further slight scapular rotation.

During the first 15 to 20 degrees of movement of the arm away from the side, our studies have convinced us that there is an initial upward movement of the head of the humerus on the glenoid. This initial movement of the humerus on the glenoid, almost imperceptible in the harmonious action of the normal joint, is revealed only by a comparison of radiograms taken under exact and standard conditions and is morphologically reflected in the elongated or pearlike shape of the glenoid cavity. Still more convincing evidence of the reality and extent of this movement of the humerus has been obtained on patients operated upon under local anesthesia in the sitting position. Under these conditions, the humerus has been observed to glide, at the initiation of abduction, upward on the tendon of the biceps for almost one-half inch. With further movement at the scapulohumeral joint, the head of the bone glides downward on the glenoid in abduction and both downward and backward in forward flexion. The validity of these conclusions is amply established by clinical experience. In the liberation of the stiff shoulder the establishment of a free pathway for the head of the humerus on the glenoid in an upward and downward direction is of primary importance, and in cases of paralysis we have been highly gratified by the results of re-education of this, the normal sequence. The prominence exhibited at the anteroinferior lip of the glenoid is a related structural arrangement. Recurrent dislocation of the shoulder joint is, as is well known, a common sequel to fracture of this lip or to tearing of the attachment of the related capsule. It is the anteroinferior part of the joint which, as abduction is increased, holds and prevents, to some extent, displacement of the humeral head.

Having discussed some of the details of motion at this joint, the next consideration is the function of the related muscles. "The primary movement which the deltoid imparts to the humerus," says Greig, "is not abduction but a drawing of the humerus directly upward." This

author has shown by an examination of the phylogeny, anatomy, innervation, and also by clinical illustrations that the muscle which arrests and changes the direction of the deltoid, is the teres minor. This muscle steadies the humeral head in the glenoid and renders the action smoother. In forward flexion the mass of the pectoral musculature is thrown into action which tends to displace the head of the humerus, a tendency which cannot be altogether overcome by the action of the teres minor and major muscles. It is here, we feel, that the long head of the biceps muscle plays its chief role. This tendon, from its course and direction, serves mechanically to guide and redirect the humeral head in its backward and downward path so that action as a whole is gracefully synchronized.

That this is the function of the long head of the biceps is borne out by clinical observations. Rupture or operative severance of the long head of the biceps is seldom attended with any functional loss of great consequence. We have always observed, however, that in these cases there is a loss of strength and rhythm which is most evident in forward flexion and especially in movements above the right angle. Undue prominence of the humeral head anteriorly is noticeable in cases of rupture or dislocation of the tendon or after the Nicola operation. This is apparent in some of the clinical histories given below and was very noticeable in the subsequent history of Case 1. The necessity of the direct attachment of the biceps to the scapula in order to restrain the head of the humerus was appreciated by Duchenne, who states: "Here are clinical facts which show the utility and also the necessity for the direct attachments of the biceps brachii to the scapula. Subjects in whom I have discovered the biceps brachii atrophied were still able to flex the forearm strongly with assistance of the brachialis anticus and the brachioradialis but, at that time, if resistance to flexion of the forearm was great, as in raising a heavy weight from the ground, by flexing the forearm on the arm, the head of the humerus was displaced from the glenoid cavity and the subject would experience, in the articulation of the scapulohumeral joint, a pulling sensation which became painful if the weight was too heavy or if the movement was too prolonged or too frequent. For the workman this was a cause of weakness or deficiency and he was unable to carry on laborious work for a long time without suffering. This fact demonstrates, therefore, that it is necessary that one of the muscles which produces flexion should, at the same time, maintain the head of the humerus against the glenoid cavity of the scapula. These conditions find themselves perfectly supplied by the long portion of the biceps brachii." In addition, there is no doubt that the long head of the biceps is a mild, medial rotator when the arm is in lateral rotation and at the side of the body.

The action of the long head of the biceps at the humerus must be regarded as both active and passive. "It is," says Codman, "the upper

end of the humerus, using the tendon and canal as a guide, which rides up and down in motions of the shoulder joint and not, as superficially thought, the long head of the biceps which plays in the groove when the forearm is moved.''

The practical importance of these observations is apparent from a study of the following six cases in which operation proved the diagnosis.

CASE 1.—L. C., male, 37 years of age. *Chief Complaint:* Pain and weakness of the left shoulder. *Present Illness:* The patient sustained his injury on Aug. 2, 1932, while lifting three-hundred-pound blocks of ice. As he lifted one block, he felt a severe pain in the front of the left shoulder. Unable to continue his work, he consulted his physician who prescribed rest and heat. After a few days' treatment, he tried to return to work, but was unable to do so because he could not lift a weight of even sixty pounds. He had been doing heavy work continually for thirteen years prior to his accident.

We first saw the patient four weeks after the injury. He complained of a dull pain over the anterior aspect of his left shoulder which was aggravated by abduction and flexion.

Physical Examination.—The patient was a well-developed and muscular man. His left shoulder appeared slightly swollen and there was generalized tenderness over its anterior aspect. The most sensitive area lay directly over and just lateral to the coracoid process.

On physical examination active abduction at the shoulder was limited to 120 degrees; flexion was free to 180 degrees. Moderate abduction and lateral rotation were associated with pain and a ''clicking'' sensation could be elicited in the region of the coracoid process. Flexion of the elbow or flexion of the shoulder against resistance caused pain in the same area. All motions of the shoulder were weak, notably abduction and flexion.

The striking finding was that moderate abduction of the arm with external rotation produced a definite snap which could be felt over the anterior aspect of the joint. A dislocation of the tendon of the long head of the biceps was suspected and operation was advised.

Operation, Dec. 9, 1932.—An incision was made over the anterior aspect of the shoulder with separation of the deltoid and pectoralis major muscles. The long head of the biceps tendon was found to be dislocated over the lesser tuberosity of the humerus. The supraspinatus tendon was ruptured partially at the margin of its attachment to the anterior border of the greater tuberosity. With the arm abducted to about 70 degrees, rotations caused the tendon of the long head of the biceps to ride backward and forward over the lesser tuberosity. On external rotation it was dislocated to the medial side of the tubercle and on inward rotation it reduced itself with an audible snap. There was slight fraying of the tendon in its intracapsular portion together with a partial tear of the origin of the tendon of the long head of the biceps from the margin of the glenoid.

Repair consisted of deepening of the bicipital groove with replacement of the tendon and reconstruction of the roof. The supraspinatus tendon was sutured with chromic catgut. The shoulder was immobilized in abduction.

Subsequent Course.—The postoperative treatment consisted of baking, massage, and exercises. After ten months, flexion and supination of the forearm were free, strong, and painless, but marked weakness of abduction and forward flexion of the shoulder persisted.

Second Operation, Oct. 6, 1933.—Under local anesthesia the old scar was excised and the capsule of the joint and the bicipital groove were opened. The distal por-

tion of the long head of the biceps was found firmly attached to the floor of the bicipital groove and its proximal intracapsular portion had disappeared, except for a few fibrous strands which were adherent to the adjacent capsule. These strands were excised and the capsule was sutured.

The postoperative treatment consisted of baking and exercises. The patient exhibited some weakness of the shoulder, especially in abduction and forward flexion. With the elbow extended, he was unable to flex the arm forward at the shoulder against any considerable resistance. The function of the forearm in flexion and supination was excellent.

Examination on April 17, 1934, showed no further snapping on abduction and external rotation but otherwise showed a failure of any important improvement in his condition. The functional result in this case was entirely unsatisfactory because of the marked weakness at the shoulder in forward flexion and abduction. In all probability this was caused by lack of fixation of the humeral head as a result of the combined lesion of the supraspinatus and the tendon of the long head of the biceps.

The patient was re-examined on Dec. 21, 1938. He had weakness and pain in the shoulder on motion and in damp weather. He complained of a cracking sensation in the shoulder on abduction and of weakness when he attempted to lift a heavy weight.

Examination at this time showed abduction to be limited to 135 degrees; forward flexion to 170 degrees. Active abduction could be carried to 80 degrees. When the arm was in full abduction and flexion, a very definite snapping could be heard and felt and this snapping was associated with pain.

CASE 2.—F. G., male, 40 years of age. *Chief Complaint:* Pain and weakness of the right shoulder. *Present Illness:* On Nov. 28, 1932, while lifting a heavy piece of ice, he had a severe pain in the anterior aspect of the right shoulder which caused immediate disability. In one month's time all of the subjective symptoms had disappeared and the patient was placed on active exercises. Two days later he again complained of pain in the front of the shoulder. The re-examination disclosed tenderness over the anterior aspect of the shoulder with pain and weakness on forward flexion and abduction. Abduction and external rotation elicited a clicking sensation in the region of the bicipital groove. Stereoscopic roentgenograms of the shoulder at this time disclosed no evidence of bony change or arthritis.

Physical Examination.—There was moderate, generalized atrophy of the muscles of the affected shoulder. Palpation revealed tenderness over the anterior aspect of the shoulder in the region of the bicipital groove. There was a distinct disturbance in the scapulohumeral rhythm on the right side. Abduction was initiated quite freely, but, when an angle of 50 degrees was reached, the shoulder hunched with pain before further abduction could be obtained. On several occasions, when moving his arm into active abduction, a marked snapping could be felt in the region of the bicipital groove. Active flexion and supination at the elbow against resistance caused pain referred to the same region.

Roentgenograms taken on March 6, with the shoulder in internal and external rotation, showed irregularity of the bone along the margin of the lesser tubercle of the right humerus. A diagnosis of dislocation of the long head of the biceps was made.

Operation, March 8, 1933.—An incision was made through the anterior portion of the deltoid muscle. The principal findings were dislocation of the tendon to the medial side of the lesser tubercle, partial tear of the adjacent attachment of the

subscapularis tendon, and a filling of the bicipital groove with scar tissue. The tendon was fixed to the floor of the bicipital groove. (Figs. 4-8.)

Subsequent Course.—The convalescence was uneventful and the patient was discharged from the hospital on March 29, 1933. On June 30, 1933, there was active

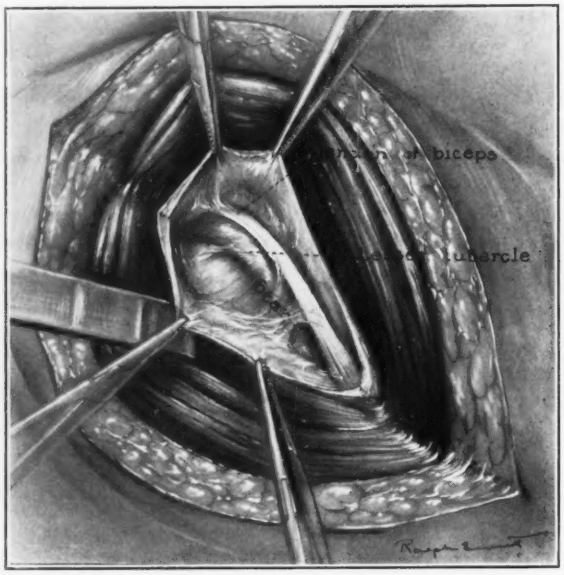


Fig. 4.—The tendon of the long head of the biceps brachii is dislocated over the lesser tuberosity of the humerus and the intertubercular groove is almost obliterated.

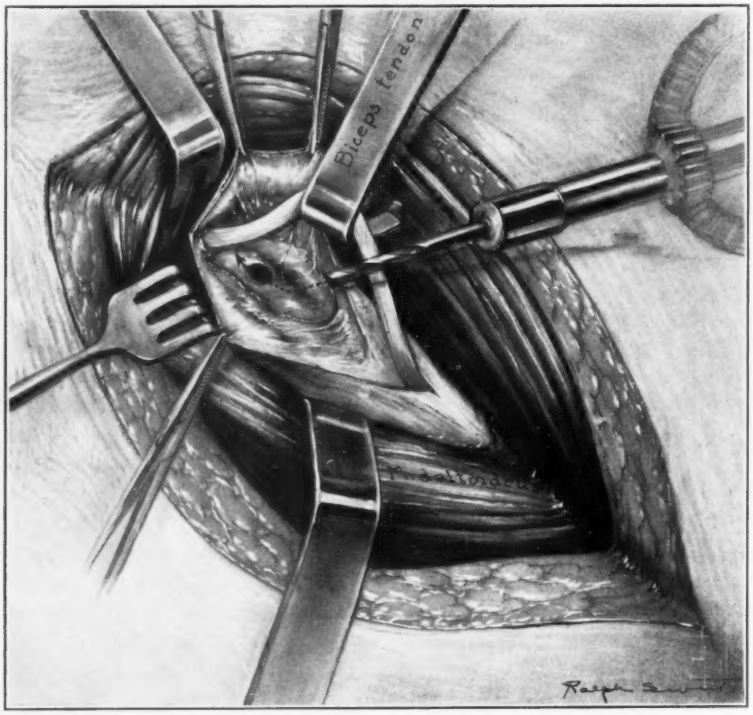


Fig. 5.—Method of placing drill holes for fixation of the tendon.

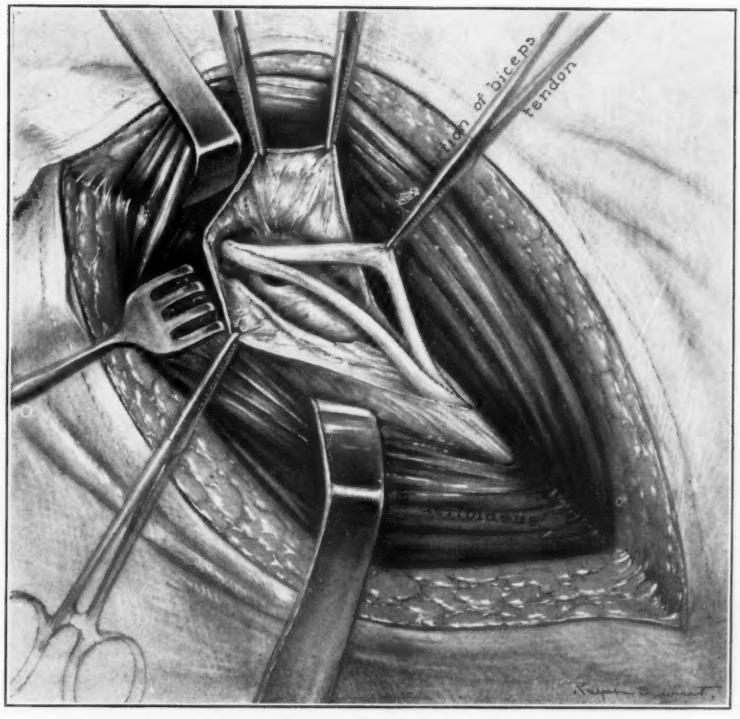


Fig. 6.—Division of the tendon preparatory to fixation in drill holes.

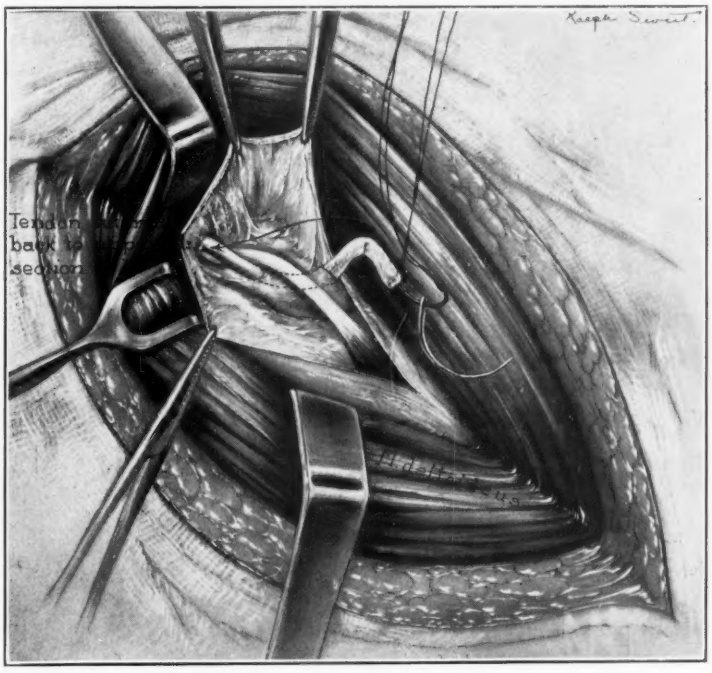


Fig. 7.—Method of fixing tendon in the bicipital groove.

abduction to 95 degrees and forward flexion to 120 degrees. External rotation was complete. The clicking sensation in the region of the bicipital groove had disappeared entirely. The patient complained of some weakness of the shoulder and of pain along the vertebral border of the scapula.

Treatment consisted of exercises, baking, and strapping across the vertebral border of the scapula. This latter fixation afforded considerable relief from the pain in this area.

Operation, Oct. 26, 1933.—The patient continued to complain of pain and tenderness at the base of the spine of the scapula.

Under local anesthesia this region was incised and the only possible source of trouble was what appeared to be some scar tissue overlying the spine of the scapula near the vertebral border. This mass was excised and the wound closed.

Convalescence following the operation was uneventful and the tenderness disappeared from the base of the spine of the scapula. The symptoms referable to slipping of the biceps tendon had disappeared entirely. Flexion and abduction of the shoulder, flexion and supination of the forearm, and the strength of the arm were quite satisfactory.

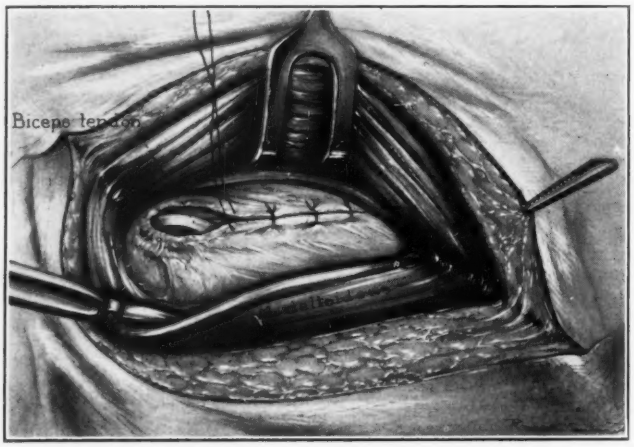


Fig. 8.—Closure of fibrous tissue sheath over the bicipital groove.

CASE 3.*—Male. *Chief Complaint:* Pain and weakness in the right shoulder. *Present Illness:* On Sept. 15, 1932, the patient fell a distance of twelve feet, landing on the palm of his right hand. He sustained a fracture of his right wrist and wrenched his right shoulder. The function of the wrist gradually returned to normal. Three months after injury, the patient still complained of dull pain, loss of strength, and a "cracking" sensation in the right shoulder joint.

Physical Examination.—The important findings were that abduction was limited to 90 degrees, forward flexion to 120 degrees, and that there was a loss of 50 per cent in external and internal rotations. Forced motions of the shoulder joint were painful. A rupture of the supraspinatus tendon was suspected.

Second Operation, Jan. 10, 1933.—An anterior incision was made separating the deltoid and pectoralis major muscles. There was no evidence of a tear of the supraspinatus muscle. The tendon of the long head of the biceps muscle was not in the intertubercular groove but was dislocated to the medial aspect of the lesser tuberosity.

*Presented through the courtesy of Dr. W. J. Cox, San Francisco, Calif., to whom we are indebted for the history and postoperative notes.

The tendon was easily replaced in its groove but redislocated on external rotation of the shoulder. The groove was deepened and the tendon held in its floor by drill holes and silk sutures.

Subsequent Course.—The patient had physical therapy for approximately five months. Examination then showed a normal range of abduction, forward flexion and rotation. The strength of the arm was satisfactory.

CASE 4.*—E. G., male, 56 years of age. *Chief Complaint:* Pain and weakness in the right shoulder. *Present Illness:* On July 27, 1933, the patient fell head first a distance of ten feet and was caught between two timbers with both arms abducted. Immediately after the accident he felt pain in both shoulders. A few days later there were pain, swelling, and ecchymosis over the anterior aspect of the right shoulder joint. On attempting to raise the arm "something seemed to fly out of place accompanied by pain and a snapping sensation which would almost knock him down." On lowering the arm to the side, "he felt something snap back into place" and the pain was relieved.

Physical Examination.—There were a small area of ecchymosis and a considerable area of tenderness over the anterior aspect of the shoulder joint. Active abduction was limited to 25 degrees and there was a slight limitation of rotation.

At this time a tentative diagnosis of injury to the short head of the biceps was made. Conservative measures were carried out for several months. On Oct. 23, 1933, the patient stated that for the past four or five days he had noticed that he had felt a sudden catch just lateral to the anterior axillary fold when he reached overhead with the right arm forward and the elbow flexed.

Examination elicited a painful "snapping" sensation in the region of the bicipital groove when the arm was externally rotated. A diagnosis of dislocation of the tendon of the long head of the biceps was made.

Operation, Oct. 24, 1933.—The operation was performed under local anesthesia. On retraction of the deltoid, the bicipital groove was readily brought into view. With abduction and external rotation the long head of the biceps tendon slipped inward over the lesser tuberosity. The bicipital groove was narrow, unusually shallow, and contained a mass of injected scar tissue. A flat cartilaginous excrescence, 1.5 cm. in size, was found loosely attached to the articular surface of the humerus immediately proximal to the lesser tubercle. On enlarging the capsular incision, the scar tissue filling the groove became continuous with the capsule and dissipated itself into the latter about 1 cm. proximal to the lesser tuberosity. After removal of the scar tissue and replacement of the tendon, the roof of the groove was reconstructed by suturing the fibrous tissue to two drill holes lying just below the lesser tuberosity. Following repair, manipulation of the arm and movement by the patient failed to displace the tendon.

Subsequent Course.—The pain had diminished and free movement of the joint was established. There was pain, however, on pressure over the region of the bicipital groove and on the extremes of abduction, external rotation at the shoulder, and on pronation and supination of the forearm. Supination was especially painful when performed against resistance.

Five months later, the patient complained of increasing pain and limitation of motion in the right shoulder. Examination showed tenderness along the course of the long head of the biceps tendon with severe pain on flexion and supination of the forearm. On abduction and external rotation, there was pain in the region of the bicipital groove. Internal and external rotations with the arm abducted to 70 degrees caused excruciating pain over the long head of the biceps.

*Presented through the courtesy of Dr. J. M. Meherin, San Francisco, Calif., to whom we are indebted for the history and postoperative notes.

The conclusion reached at this time was that the long head of the biceps tendon had redislocated from the bicipital groove and that further surgery would be necessary.

The patient was re-examined on April 12, 1934. His chief complaint was of pain, soreness in the shoulder, marked weakness and limitation of motion. The snapping sensation was entirely relieved. The pain in the shoulder was more severe than it had been when this patient was seen about three months before.

There was tenderness over the anterior aspect of the shoulder, particularly in the region of the bicipital groove. Attempts to carry out abduction and forward flexion were painful and limited. There was no snapping elicited on abduction with external rotation. Flexion and supination of the forearm, particularly the latter, were painful. The structures in the region of the bicipital groove were exceedingly tender.

Second Operation, April 20, 1934.—The old scar was excised and the bicipital groove was exposed. The deltoid was retracted laterally and, with rotation of the humerus, no definite injury to the supraspinatus or subscapularis was seen. The scar tissue over the groove was incised and the flattened tendon of the long head of the biceps was found displaced in front of the lesser tuberosity. The tendon above and below the groove was apparently normal without evidence of injury, nor was there fraying of its margins.

The reconstructive procedure consisted of removal of the origin of the long head of the biceps tendon from the glenoid and fixation of the tendon to the bicipital groove.

At the last examination, on Dec. 18, 1935, the patient stated that he was unable to do any work.

The examination showed that there was more atrophy of the muscles of the right shoulder than of the left shoulder. He complained of some tenderness over the bicipital groove and the tip of the coracoid process. Abduction and forward flexion of the shoulder could be carried out to 125 degrees and 180 degrees respectively. Rotations were limited to three-fourths of the normal range. All movements of the elbow were normal. The examination of the left shoulder showed complete range of active motion with the exception of internal rotation which was carried out to three-fourths of the normal range. All movements of the left elbow were free and painless.

CASE 5.—P. M., male, 46 years of age. *Chief Complaint:* Pain with inability to abduct the right arm. *Present Illness:* In January, 1935, the patient fell from a hay stack. He was holding a pitchfork with both hands and recollects that he landed on his hands, turned over, and struck heavily on his head and right shoulder. The right shoulder was severely "twisted" and the patient stated that he was unconscious for a short time. After the accident, he was unable to elevate the right arm. He did not seek professional advice but applied heat and liniments to the shoulder to alleviate pain. Some weeks later, on rotating the arm inward and then outward, he often felt a "pop" which, on occasions, was so loud that it could be heard across the room. Since his injury, he had suffered continually from an ache in the shoulder.

Physical Examination.—The patient was first examined on Jan. 14, 1936, one years after injury. Physically, he was a well-developed and well-nourished man. He held the right shoulder in a drooping position, but did not exhibit any obvious muscular atrophy nor any change in muscular contour. With the arm held in internal rotation, he was able to reach the complete range of flexion in the shoulder. On lowering the arm, when it reached an angle of 90 degrees, there was a sudden pain and a "grating" sensation and the arm fell to the side. Active abduction was limited to 30 degrees but could be increased passively to 90 degrees, at which

point the patient complained of pain referred to the insertion of the deltoid muscle. Flexion of the forearm against resistance both in pronation and in supination was equal on both sides and there was no alteration in the contour of the biceps. On palpation there was a little tenderness over the upper end of the intertubercular sulcus. On lateral rotation of the arm a "snapping" sensation could be distinctly elicited through the substance of the deltoid muscle in the region of the lesser tuberosity of the humerus. The long head of the biceps muscle could be palpated in its lower part but exhibited no abnormality of position.

Operation, Jan. 16, 1936.—The patient was placed in the sitting position with the arm slightly abducted. Under local anesthesia the deltoid muscle was split and the shoulder joint revealed. The long head of the biceps was found dislocated from its groove and lying medial to the lesser tuberosity. The supraspinatus tendon was intact. The long tendon of the biceps was severed and implanted into the greater tuberosity in such a manner that its normal direction of action was maintained and the proximal part of the tendon was excised. After closure of the wound, the arm was put up in abduction.

Subsequent Course.—On Feb. 23 the cast was discarded and baking, massage, and exercises were commenced. On March 27 examination showed some weakness on abduction, but there was no pain and no evidence of slipping of the tendon. A month later there was considerable improvement. Abduction and forward flexion were stronger and there was no pain.

The patient was re-examined on Nov. 22, 1938, two years later, at which time he did not experience any pain. The snapping over the rim of the bicipital groove had disappeared entirely. His chief complaint was weakness and limitation of abduction. He was able to carry on with his occupation as a farmer. He stated that his shoulder was "good enough" and that he did not wish to submit to any further surgery.

Examination disclosed some tenderness over the anterior aspect of the upper end of the humerus. In active motion of the right upper extremity he hunched his shoulder and there was a distinct disturbance in the scapulohumeral rhythm. Abduction was carried out largely by use of the scapula on the chest wall. The arm could be elevated passively to 30 degrees beyond the right angle and the patient could hold the arm in this position. In other words, range of passive motion was greater than active motion at the scapulohumeral joint.

From the present clinical findings, we feel that this patient may have had a partial rupture of the deep fibers of the insertion of the supraspinatus, a fact which could not be detected at the time of operation unless incisions had been made through the outer, intact portion of the tendon.

CASE 6.—H. S., male, 39 years of age. *Chief Complaint:* Pain and weakness in the right shoulder. *Present Illness:* The patient was a warehouseman. On April 9, 1938, he fell from a ladder striking his right shoulder against the edge of a table. He had severe pain in his right shoulder and arm immediately after the injury. The pain persisted and two days later he consulted his physician who prescribed physical therapy. At this time his shoulder was noticeably swollen and very painful.

Physical Examination.—The patient was examined by us on April 22, 1938, thirteen days after his injury. He held his right arm rigidly at his side and when attempts were made to move his shoulder there were spasm of the muscles and severe pain. Because of this pain, he was unable to abduct and flex his arm. There were marked tenderness over the bicipital groove, visible swelling in this region, and loss of tone of the belly of the biceps muscle.

With assistance the patient was able to initiate abduction and flexion and to show contraction of the deltoid muscle. Active, external and internal rotations were impossible because of pain and spasm.

Roentgenograms of the right shoulder taken on April 11, 1938, showed no evidence of injury to the bone.

Conservative measures did not relieve him and operation was advised.

Operation, May 21, 1938.—An incision was made over the anterior surface of the right shoulder with separation of the fibers of the deltoid. The musculotendinous cuff involving the supraspinatus and infraspinatus muscles was found to be torn and retracted from its insertion. The tendon of the biceps was displaced from the bicipital groove and carried forward and inward with the torn musculotendinous cuff. Further dissection showed the tendon of the biceps wedged between the anterior and upper portion of the rim of the glenoid and the head of the humerus. This displacement prevented internal rotation of the humerus.

The falciform edge of the musculotendinous cuff was pulled down and fixed by strong silk sutures on the upper margin of the lateral surface of the humerus after the method of Wilson. Following reattachment of the cuff, the tendon of the biceps was readily replaced in the bicipital groove. The fibrous roof of the bicipital groove was sutured to the fascia and periosteum at its outer margin. The wound was closed with fine silk and a plaster of Paris spica was applied, holding the arm in 60 degrees abduction.

Subsequent Course.—Immobilization was continued for six weeks, when active exercises in abduction and rotations were begun. On Aug. 24, 1938, he showed a satisfactory improvement in function, although he complained of some pain and a "clicking" sensation on passive abduction and external rotation. He could abduct his arm actively to 150 degrees and rotate internally and externally to 50 per cent of normal.

The patient reported daily for physical therapy and monthly examinations revealed a gradual improvement in motions of the shoulder and in strength. He resumed his normal occupation in November, 1938. At the time of the last examination, Dec. 8, 1938, he stated that hard work often gave rise to pain in his right shoulder. He was instructed to continue with his work so that motion and strength could be increased in the shoulder.

ETIOLOGY

The clinical histories and operative findings in these cases are of special interest in relation to the observations made by Meyer on the cadaver as to the etiology of dislocation of the long head of the biceps brachii muscle. In this author's opinion displacement of the tendon is primarily the result of attrition of the joint capsule proximal to its attachments at the lesser tubercle. In his view the process is a gradual one and is supported by evidence of intermediate or transitional stages. Meyer is strongly, if not vehemently, opposed to the idea of trauma as an etiologic factor.

It is possible, in the cases outlined above, that the laborious occupations followed by these patients may have led to attrition of capsular attachments and that the accident simply produced the final displacement of the tendon. On the other hand, we cannot ignore the fact that in each of these cases there was a definite and severe accident in which wrenching of the shoulder joint was a prominent feature. In addition, and what we consider of the greatest significance, are the fairly uniform findings at operation. There were scar tissue, rupture of the fibrous roof

and capsular attachments in the region of the tuberosities, and, in some, an attendant lesion of the supraspinatus or subscapularis tendon, all of which show extensive local injury recent in origin. In the light of these findings, while recognizing from Meyer's work and from similar material which we have inspected the importance of attrition, we must conclude that in these cases trauma was the most significant factor in the production of the dislocation. Therefore, it is probably advisable to distinguish etiologically between acute traumatic dislocations and the chronic, progressive dislocations described by Meyer. Unfortunately, we have no clinical histories in Meyer's cases.

It would appear to us, after a study of these cases, that forcible lateral rotation of the arm was the principal cause of dislocation.

DIAGNOSIS AND DISCUSSION

An analysis of the foregoing cases will demonstrate that in each instance the patient gave a history, up to the time of his accident, of having been able to do work which involved strenuous use of his upper extremities. In other words, there was no history of previous complaint. In all cases, following upon a severe wrenching injury to the shoulder, there was complete and immediate disability. In four of the cases a snapping sensation was noted by the patient or by his physician a few days after the accident; in the fifth case, at the end of several months.

The outstanding clinical findings were: (1) swelling of the anterior aspect of the shoulder directly following the accident, (2) tenderness over the region of the long head of the biceps and the bicipital groove, and (3) limitation of motion and loss of strength in forward flexion and abduction. Pronation and supination of the forearm against resistance were painful, the pain being referred to the region of the bicipital groove. The outstanding feature, common to all six patients, was the "clicking" in the region of the bicipital groove which could be seen and felt on moderate abduction and lateral rotation of the arm.

Abduction with the arm laterally rotated is characteristically limited, and, if carried out passively, painful resistance is usually encountered. In the less acute stages, if first medially rotated, the arm may be carried above the head either actively or passively. This is more readily accomplished in flexion than in abduction. These movements are obviously permitted by the preliminary reduction of the dislocated tendon in the medial rotation. As the arm is carried from above the head to the side, when it reaches a point somewhat below the right angle, an audible or palpable snap may be evident which is associated with redislocation of the tendon. For reasons already discussed, redislocation occurs most readily in lateral rotation at about 45 degrees of abduction, and reduction is accomplished most easily by medial rotation with the arm at the side.

DIFFERENTIAL DIAGNOSIS

The conditions most likely to be confused with dislocation of the tendon of the long head of the biceps are: (1) rupture of the supraspinatus, (2) subcoracoid bursitis, (3) tenosynovitis of the biceps tendon, (4) snapping shoulder, (5) injury to the muscles which have their insertions on the margins of the bicipital groove, and (6) referred pain from irritation of the cervical nerve roots.

1. Rupture of the supraspinatus tendon may be very difficult of differentiation and it may occur simultaneously, as illustrated in Case 1. Both injuries occur in patients employed in laborious occupations and are followed by severe pain and loss of power in the arm, especially in forward flexion and abduction at the shoulder. There is tenderness around the anterior aspect of the shoulder in both conditions. In dislocation of the tendon of the biceps, however, the tenderness is more evident in the region of the bicipital groove than over the greater tuberosity of the humerus. In both conditions there is a disturbance of the scapulohumeral rhythm, but in our cases we did not find the definite jog and soft crepitus as the tuberosity disappears beneath the acromial process which we associate with lesions of the supraspinatus tendon. In dislocation of the tendon the disturbance in rhythm is often accompanied by a distinct snapping sensation on abduction and external rotation of the arm. Pain is also present on flexion of the elbow and supination of the forearm against resistance.

2. Subcoracoid Bursitis.—Codman stated in his earlier papers that he made the diagnosis of subcoracoid bursitis as if it were a distinctly separate thing from subacromial bursitis. Later he described the subcoracoid bursa as only a part of the subacromial bursa. He assumed, however, that the subcoracoid portion might be inflamed without involvement of the entire bursa. In such cases there is tenderness in the region of the lesser tuberosity of the humerus with crepitus and limitation of rotation of the shoulder. In one patient these symptoms were elicited and a diagnosis was made of partial dislocation of the biceps tendon from its groove. At operation no dislocation was found, but there was a marked thickening of the subcoracoad portion of the subacromial bursa. The operation was performed under local anesthesia, and we observed that when the arm was actively abducted and rotated externally this thickened portion impinged upon the common tendon of the origin of the short head of the biceps and of the coracobrachialis. This produced pain and a snapping sensation, symptoms which were exactly identical with those the patient had described prior to the operation.

The removal of this thickened portion gave complete relief.

3. Tenosynovitis of the tendon of the long head of the biceps may occur and in some cases crepitus in the region of the bicipital groove

with limitation of abduction and external rotation led to difficulty in diagnosis. At operation scar tissue was found involving the subacromial bursa with adhesions between its walls, and there were also adhesions between the tendon of the biceps and its synovial sheath. Our diagnosis was subacromial bursitis and tenosynovitis of the biceps tendon following direct trauma to the anterior aspect of the shoulder. This condition will be discussed at greater length in a future report.

4. Snapping shoulder has been described in cases in which a definite audible snap occurs with abduction and external rotation of the arm. We have encountered this condition only in cases of dislocation of the tendon of the long head of the biceps and in the case of subcoracoid bursitis described above.

5. Injury to the insertion of muscles at the bicipital groove is accompanied by pain and tenderness over the site of insertion with spasm of the affected muscle. These ruptures are usually the result of direct violence and are accompanied by sharp pain. There also may be a loud snap. A break in continuity of the tendon can be ascertained with loss of function.

6. We need only refer briefly to irritations of the nerve roots which may cause pain in the shoulder. The most important of these is arthritic involvement of the lower cervical spine. This condition is a frequent cause of pain in the shoulder, but a careful examination will show pain and limitation of motion of the involved cervical spine. Roentgenograms are also helpful in diagnosis. Referred pain caused by the pressure of a cervical rib or pain caused by a contracted scalenus anticus muscle (the Naffziger syndrome) may be confusing in diagnosis. It has been our experience, however, that in these conditions we do not find well-defined tenderness over the shoulder, limitation of motion, and spasm of the muscles which move the scapulohumeral joint.

THE OPERATIVE PROCEDURE

Operation is indicated for traumatic dislocation of the tendon of the long head of the biceps. We wish to emphasize here that the position of the patient for operation is important for adequate exposure of the structures of the joint. For a number of years it has been our practice to operate on children in the sitting position for arthrodesis of the shoulder. Only in this way can one estimate with accuracy the degree of abduction in the scapulohumeral joint. Within the past seven years, however, we have used this position for operations on rupture of the supraspinatus tendon, for calcified deposits in the subacromial bursa, and for complete exposure of the joint by the sabre-cut incision of Codman. With the patient in this position, the surgeon has access to all aspects of the shoulder.

In this procedure we have found the use of 1 per cent novocain to be almost ideal for exploration of the shoulder joint. It has the advantage

of permitting the patient to move his shoulder actively during the operation.

For exposure of the bicipital groove, the incision described by Codman should be used. With the arm at the side, the elbow flexed, and the forearm pointing straight forward, an anterior incision is made downward from just below the acromioclavicular joint. The fibers of the deltoid muscle are separated with exposure of the bicipital groove and the tuberosities of the humerus. By extension and rotation one can expose the major portion of the insertion of the supraspinatus, infraspinatus, teres minor, and subscapularis muscles. In our opinion the incision with separation of the pectoralis major and deltoid muscles, as used in some of our cases, is unnecessarily radical and need not be employed.

With dislocation of the tendon from its groove, the surgeon has the choice of division of the tendon at its origin from the glenoid with fixation of the tendon to the floor of the bicipital groove, or replacement of the tendon with reconstruction of the fibrous roof to prevent its redislocation. The first procedure is much the simpler. It was employed in Case 2, in which there was no associated injury to the supraspinatus tendon. A good functional result was obtained. The patient could flex his elbow, supinate the forearm, and flex at the shoulder against considerable resistance. Case 1 demonstrated, however, the importance of preservation of the function of the long head of the biceps when there is an associated lesion of the supraspinatus. In this patient fixation of the biceps tendon to its groove and the resulting disappearance of its intracapsular portion led to an unstable shoulder and an unsatisfactory result. The association of these two lesions would seem a definite indication for adequate repair of the supraspinatus and an effort to preserve the long head of the biceps by plastic repair of the roof of the bicipital groove. In a case of this type the use of fascia would seem appropriate.

SUMMARY

1. Six cases of dislocation of the biceps tendon resulting from injury are described.

2. In all cases the injury was followed by pain and swelling on the anterior aspect of the joint and complete disability of the affected shoulder.

3. The patients complained of pain with weakness and limitation of motion in forward flexion and abduction of the shoulder and pain on forced supination of the forearm against resistance with the elbow held in flexion.

4. The principal findings were swelling over the anterior aspect of the shoulder, tenderness, most pronounced over the bicipital groove, and a definite snapping sensation on abduction and external rotation of the shoulder.

5. In all cases operation disclosed a displacement of the tendon over the lesser tuberosity.

6. The best approach is obtained by the anterior muscle-splitting incision described by Codman.

7. In our opinion, local anesthesia and the sitting position greatly facilitate the operation.

8. No rules can be formulated as to the best procedure to be adopted in cases of dislocation of the tendon of the long head of the biceps. In uncomplicated dislocation it is highly probable that the quickest and best restoration of function can be obtained by fixation of the tendon in the bicipital groove. When other lesions occur simultaneously, an adequate result may be obtained by replacement of the tendon and repair by fascia of the roof of its groove.

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A BLIND METHOD OF NAILING FRACTURED FEMORAL NECKS

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AFTER a fracture of the femoral neck has been satisfactorily reduced and one wishes to fix the fractured fragments by indirect nailing, six unknown factors need to be solved. These unknowns are: (1) the place on the thigh where the skin incision is to be made; (2) the point on the long axis of the femur where the nail is to be started; (3) the vertical angle to be assumed by the nail and driver; (4) the length of nail to be used; (5) the horizontal angle to be assumed by the driver; (6) the point on the short axis of the femur where the nail should be started.

The protractor and measure described in this article is efficient, simple to use, and determines very quickly the first four unknowns. It does this without interfering with the taking of lateral x-rays, as described by Johnson, whose method I believe to be the most accurate and simplest of any so far described. The remaining two unknowns are solved with no trouble if one bears in mind certain anatomical facts.

In Fig. 1 the protractor and measure are illustrated. The measure consists of a metal plate (*D*), the leading edge of which is ruled off by small holes to designate quarter inches and larger holes inches. A protractor which slides (*C*) is mounted on the metal plate. A metal arrow (*B*), supporting two rests (*A*), is attached to the protractor. The protractor and arrow can be locked in any desired position by thumb nuts.

After reduction is thought to have been obtained and when the anteroposterior x-ray is taken, the metal plate is held against the thigh in the region of the trochanter (Fig. 2). The plate should be on or as near as possible the same horizontal plane as the trochanter. With gentian violet or some other suitable marking material, the upper and lower limits of the plate are marked on the skin of the thigh. This is done in order to insure the accurate replacing of the plate after it has been sterilized.

The four unknowns are determined from the image of the metal plate as it shows in the anteroposterior x-ray. In order to facilitate the determinations an ordinary celluloid rule and protractor are joined by a small winged nut (Fig. 3*A*). After the known factors have been determined from the x-ray, they are transferred to the sterilized plate and protractor as they are again held against the thigh in the same position as when the anteroposterior x-ray was taken.

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DETERMINATION OF UNKNOWNS

The straight edge of the celluloid protractor is placed along the leading edge of the metal plate as it shows in the x-ray. The rule is swung so that its center passes through the axis of the femoral neck (Fig. 3*B*). The holes in the metal plate show in the x-ray. The point where the rule crosses these holes is noted. This same point on the metal plate as

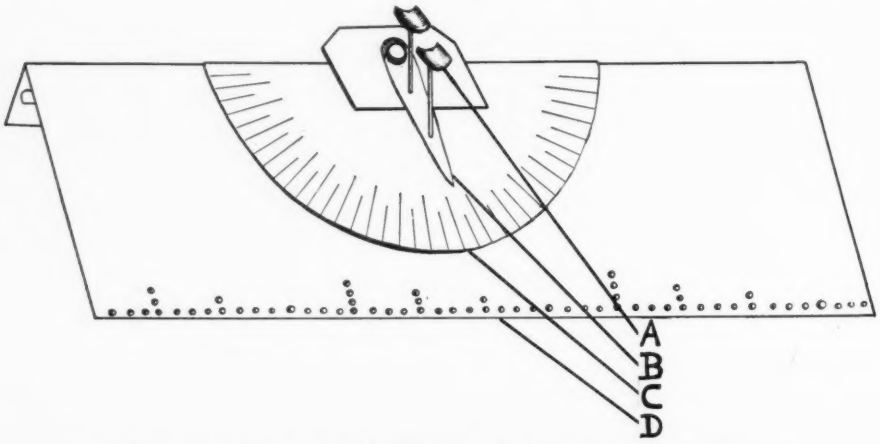

Fig. 1.—Illustration of the assembled metal protractor and measure.

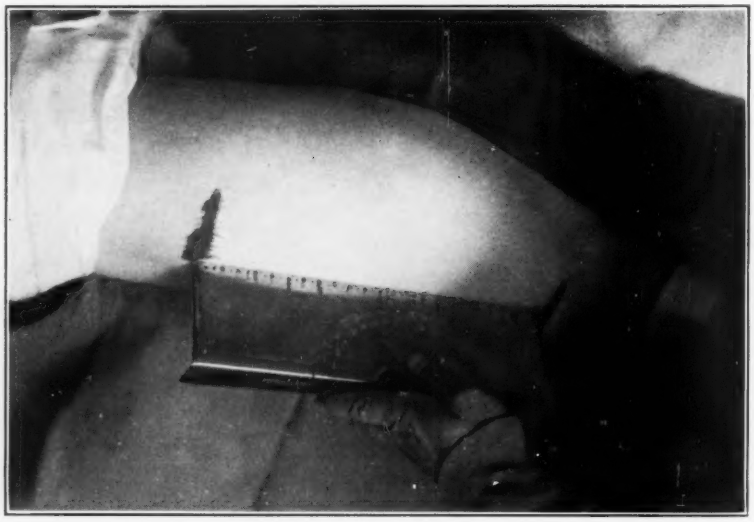

Fig. 2.—Showing the protractor and measure held against the thigh when the post-reduction anteroposterior x-ray is taken.

it is held against the thigh determines the place where the skin incision is to be made, solving the first unknown. The angle made by the center of the celluloid rule as it crosses the protractor is noted. The arrow on the metal protractor is swung to this same angle and locked. The protractor is slid on the plate so that the arrow designates the point where it has been determined the nail is to be started on the skin of the

thigh. The line of the arrow when carried to the femur determines the point on its long axis where the nail should be started, solving the second unknown. The vertical angle to be assumed by the nail and driver is the same as the angle noted where the celluloid rule crosses the protractor, thus solving the third unknown. Using the celluloid rule, the distance from the femoral head to the lateral surface of the trochanter is measured on the x-ray. This measurement is transfered

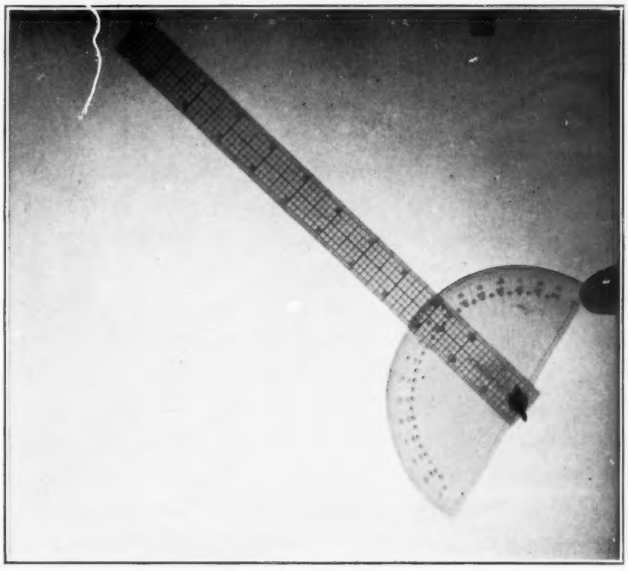

Fig. 3 *A*.—Illustration of the celluloid rule and protractor which facilitates the determination of unknown factors.

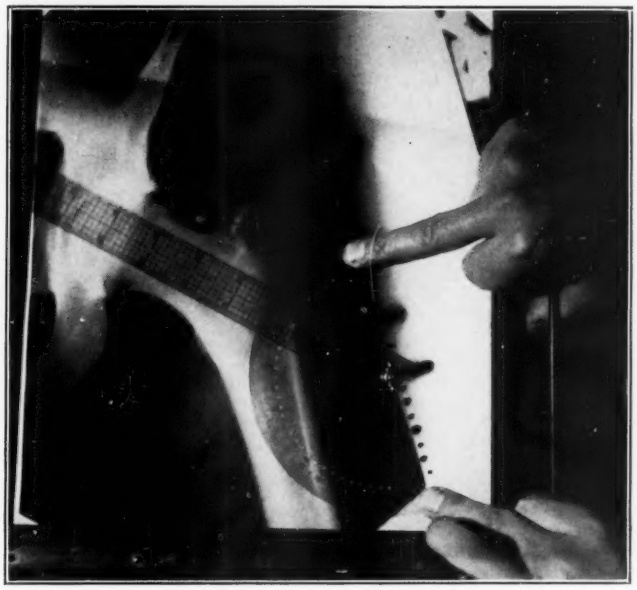

Fig. 3 *B*.—An anteroposterior x-ray of the hip, showing the image of the metal plate from which the unknown factors are obtained.

to the image of the metal rule, thereby correcting the x-ray measurement and giving the true length of the femoral neck, for the metal rule will enlarge in the same proportion as the bone. The length of the nail to be used is thus determined, solving the fourth unknown.

The two remaining unsolved factors are: (1) the horizontal angle to be assumed by the nail and driver; (2) the point on the short axis of the femur where the nail is to be started. The first is readily determined if one bears in mind that the normal degree of anteversion of the femoral neck is about 20° with the femur in a neutral rotatory position. If, on clinical examination, the knee is in a neutral rotatory plane, the nail and driver must be tilted 20° anteriorly from the horizontal. If the knee is in any other degree of rotation, the nail and driver will have to be changed in the horizontal plane, depending on the degree of change noted at the knee. The second unknown is easily determined for we know that the femoral neck comes off the anterior four-fifths of the medial surface of the femoral shaft. The nail must be started slightly anterior to the center of the short axis of the femoral shaft.

This method of determining the unknowns for blind nailing of femoral neck fractures has proved very satisfactory. It is not so foolproof that one should not check the progress of the nail as it is being inserted. In all cases in which this instrument has been used, one set of x-rays in both planes has been necessary to check the nail after it has been started. If this method is used, it is necessary that the affected leg be held in the same position between the first anteroposterior x-ray and when the nail is started. A careful assistant or a mechanical leg holder will accomplish this task.

THE USE OF ZINC PEROXIDE IN THE TREATMENT OF DIABETIC GANGRENE OF THE LOWER EXTREMITIES

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GANGRENE of the toe, foot, or leg is a frequent complication in diabetes and often has serious consequences because of its threat to the loss of limb or the loss of life.

It is well known that there is an underlying arterial sclerosis, and in some cases without any injury to the skin surface the arteries become plugged, collateral circulation is inadequate, and the area supplied by these vessels dies from loss of circulation. This results in a dry gangrene with little or no evidence of inflammation in the early stages of the process. In the later stages, however, when a line of demarcation has developed between the living and the dead tissue, a break in the continuity of the skin surface occurs, and the resulting wound becomes a portal through which contaminating bacteria may enter and produce an infection which changes profoundly the course of events, for the dry gangrene now becomes a wet gangrene.

However, in the majority of cases of diabetic gangrene of the extremities, the process is initiated by a break in the skin. This frequently starts around a toenail, where there has been undue pressure from a shoe. Occasionally it begins with a trivial wound inflicted when the patient has been going around with bare feet and has picked up a splinter or pin or has stubbed the toe with resulting abrasion. In some cases, there has been a crack in the skin from a fungus infection, occurring usually between the fourth and fifth toes. Sometimes, there has been a pre-existing corn or bunion which has been pared by the patient to the point where blood was drawn from the surface. At times, this has occurred following treatment by a chiropodist or a well-meaning member of the family.

Whenever there is a break in the skin, a portal of entry is immediately opened for the entrance of contaminating organisms, which frequently find themselves in an environment in which they can gain a foothold and multiply. Infection is favored because the tissues are poor in blood supply due to the underlying arterial sclerosis and they contain a higher percentage of sugar than is present in normal tissues. In the great majority of normal individuals, when contaminating bacteria get into a wound, no infection follows because the defensive forces of the body are promptly brought into play and the organisms are not able to gain a foothold. In the relatively few cases in which infections develop in normal people, usually only one species of bacteria can be cultivated. In

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diabetic individuals, under the same circumstances, it is much easier for organisms to gain a foothold, and a number of species may be cultivated when infections develop.

When an infection becomes established with multiple species, these organisms frequently have a certain synergistic action which they cannot perform in pure culture. Saphrophytic organisms, particularly, multiply on tissues which are dead or dying or in which the blood supply is compromised. Then, there is a great likelihood of extensive and massive death of tissue, which we call wet gangrene.

In the foot, as in the hand, there are anatomic spaces between the muscles and around the tendons in which infection may and usually does spread deep beneath the surface. The extent of such spread is often difficult to determine from any surface signs. Not infrequently, gas gangrene organisms travel up, through, and along a deep muscle to the popliteal space without showing any signs of crepitation or even swelling of the superficial tissues. Only vague pain or deep tenderness may lead one to suspect this insidious spread. Bones and joints are often invaded early with certain of these organisms, and, when such invasion occurs, the infection becomes locked off in remote nooks and crannies which are frequently missed and cannot be easily exposed.

Among the organisms frequently found in diabetic gangrene are hemolytic streptococci and staphylococci and, likewise, anaerobic or microaerophilic organisms, particularly the organisms of gas gangrene, the anaerobic nonhemolytic streptococci, and the microaerophilic streptococci. Likewise, numerous gram-negative bacilli, both aerobic and anaerobic, including *B. coli* and the pyocyaneus and proteus groups of bacteria, are found.

In the early stages of both the dry and the wet forms of gangrene, the presence of organisms and their nature can be determined by cultivation of the surface exudates. In the later stages, however, when the organisms have invaded deeply and, particularly, if the wounds or the margins of gangrene are extensive, it is necessary to take cultures from many different parts of the lesion in order to determine the organisms with which one has to deal. It is unlikely that there are any organisms in the depths that are not on the surface, but frequently organisms are present on the surface which have not invaded deeply at the time of the culture.

It is well known that bacteria differ very greatly in their power to invade. The hemolytic streptococci, the hemolytic *staphylococcus aureus* and the *B. welchii* often spread extensively and rapidly. The anaerobic streptococci, the nonhemolytic aerobic streptococci and staphylococci, and *B. coli* form an intermediate group as far as invasion is concerned. *B. proteus, B. pyocyaneus,* and the saprophytic organisms usually limit their activity to the surface or to tissues which are already dead. The most important organisms in this entire list of the flora in gangrenous

wounds are the hemolytic streptococcus, *B. welchii,* and the anaerobic streptococci. The hemolytic *Staphylococcus aureus* runs a close second to this group.

Obviously, in the care of such conditions, prophylaxis is of extreme importance. If the activity of these organisms can be nullified as soon as they contaminate the wound, or in the case of dry gangrene before the solution of continuity of the skin between living and dead occurs, the organisms will not gain a foothold and infection will not occur.

However, in most instances the infection has already gained a foothold when the patient presents himself or herself to the surgeon. Invasion for a certain distance has already taken place. There may be a diffuse cellulitis, extensive gangrene of the part, or both.

The necessary treatment depends, to a large extent, upon the rapidity of the spread of the infection and the evidence of compromise of the circulation, as determined by oscillometric and surface temperature readings. The surface temperature readings must take into account the increased heat which occurs from the inflammatory process and any rise of general body temperature must also be discounted. In some cases where the circulation is fair, the evidence of general intoxication slight, the limits of local infection narrow, conservative measures may be, and usually are, advisable. However, it is of the utmost importance for the surgeon to determine by bacteriologic examination, aerobically and anaerobically, the number and nature of the various species of bacteria which are present, and further contamination by other species must be prevented. When these organisms have been determined, appropriate medication should be applied, which not only minimizes or nullifies the growth and activity of these organisms but in no way injures the tissues.

It has been found that Dakin's solution and other hypochlorites do not act favorably on tissues which have impaired circulation. While they may be able to liquefy the slough and to that extent remove some of the pabulum on which bacteria thrive, they inhibit the development of granulation tissue and thus delay the reparative process, which is likewise a barrier against bacterial invasion. Other antiseptics, in general, injure the tissue as much as or more than they do the infecting bacteria.

A substance has recently been found, however, which markedly inhibits the growth of the hemolytic streptococci and all anaerobic bacteria, including the organisms causing gas gangrene and the anaerobic streptococci. It neutralizes or inactivates the toxins formed by these organisms and at the same time encourages the reparative process by the stimulation of granulation tissue. This new substance is zinc peroxide, a white powder which is not chemically pure, which contains between 50 and 60 per cent ZnO_2, some ZnO, $ZnCO_3$, and ZnOH.

The white powder should be suspended in sterile distilled water and applied to every part of the wound surface. The wound should then be covered with fine meshed gauze soaked in the same material. This should

be covered with compresses saturated with sterile distilled water and these compresses in turn covered with vaseline gauze or some other impermeable material to prevent evaporation. The dressing should be changed every twenty-four hours, the old material irrigated off, and a new dressing applied. It is important to use material which has been proved to be effective in the production of oxygen, and it is essential that adequate contact be obtained between the zinc peroxide suspension and every part of the wound surface.

This material, when suspended in water or moistened by body fluids, slowly gives off oxygen, producing a highly oxygenated environment wherever it comes in contact. Likewise, it forms hydrogen peroxide and these two properties result in the inhibition or death of all anaerobic organisms and certain aerobic organisms which are susceptible to the peroxides, notably the hemolytic streptococcus and the pneumococcus. There is some inhibition also to the growth of the staphylococci, but not to the same extent.

There is a limited field of usefulness for this material in the stages of infection in which the patient generally comes to the hospital. It would be useful if it were available at the time of the initiation of the process with the first break in the skin. If patients come to the doctor at this stage, it should be used as a prophylactic against infection. However, if infection has invaded deeply into the tissues so that it is not possible to get contact, zinc peroxide cannot be expected to stop the spread of the process without a preliminary operative procedure.

In anticipation of operation, no matter at what level the operation is to be done, time should be given, unless otherwise contraindicated, for the determination of the bacterial flora. As mentioned above, all of the organisms found on the surface are potential invaders in diabetic patients and may be assumed to be passing up along the lymphatics or directly through tissues to higher levels, even as high as the line of amputation.

The organisms most invasive will almost invariably reach the highest level. It must be assumed that these are present and it must be realized that if hemolytic streptococci, anaerobic streptococci, the gas gangrene organisms, or hemolytic *Staphylococcus aureus* are present, evidences of their activity must be looked for at the site of amputation and appropriate measures taken to prevent their multiplication after amputation. When the amputation is done, cultures should be taken from the stump, particularly in the region of the blood vessels and lymphatics, so that within forty-eight hours it can be definitely proved whether or not these organisms are present.

When the original cultures from the surface reveal gas gangrene organisms and hemolytic streptococci, the wound should be left open until the cultures taken at the amputation site have been reported. After forty-eight hours, if the cultures are negative for these organisms and

there is no clinical evidence of infection in the wound, a secondary closure may be done with assurance of primary healing. If these organisms are found at the site of amputation, the wound should not be closed, but should be flushed with zinc peroxide and treated daily with this material until the organisms have disappeared from the wound and all signs of infection have ceased. Then, secondary closure may be contemplated. If hemolytic streptococci or the gas gangrene organisms have not been found at the initial culture, the wound at the amputation site may be closed. However, if the cultures which are taken from the gangrene at the time of operation or at the level of amputation yield hemolytic *Staphylococcus aureus* or anaerobic streptococci, the case should be very closely observed. The wound should be inspected daily, and if there is the slightest indication of infection, it should be opened. If hemolytic *Staphylococcus aureus* is present, the wound should be dressed with potent staphylococcus bacteriophage. If anaerobic streptococci should be present, it should be dressed with zinc peroxide. If none of the four malignant types of organisms described above is found in the initial cultures, the amputation wound may be closed with every expectation of primary healing. Still, the case should be watched carefully for any general symptoms of infection, and, if the wound is not perfectly comfortable, it should be examined and appropriate measures taken.

The use of sulfanilamide is indicated in addition to the zinc peroxide if hemolytic streptococci are present. Its efficacy with other organisms is questionable. There is some evidence that sulfapyridine has a somewhat wider application, but our data are not yet sufficient to warrant any definite statements in this report.

SUMMARY

I have attempted to show how important bacteriologic studies are in connection with diabetic gangrene. Both aerobic and anaerobic cultures should be taken at the earliest possible time from several areas of the lesion when it is first presented to the surgeon, so that he may know as soon as possible with what organisms he has to deal.

Infections are usually due to a mixture of organisms, and, in the diabetic, the synergistic action of these bacteria is more likely to produce serious consequences than in normal individuals.

Zinc peroxide is the most effective antiseptic which we know of to minimize the possibility of the establishment of infection at the time of the primary injury or at the time of the first solution of continuity of the surface protection. It may, likewise, be employed during the period of preparation before operation, providing organisms are present which are susceptible to it, such as the hemolytic streptococci and all types of anaerobic organisms.

If these organisms are present at the site of amputation, it should be used as a prophylactic to prevent the development of infection in the

stump; or, if infection develops in the stump, it should be used at the earliest possible time in order to control such infection. If reamputation is necessary, it should be used immediately to prevent establishment of infection in the second operative site.

The method of employment of zinc peroxide has been given in detail.

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TOTAL CYSTECTOMY FOR CANCER

A Critical Review

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INTRODUCTION

COMPLETE removal of the bladder has become established as an important and valuable surgical procedure. It has its particular value in the treatment of certain cancers of the bladder, which are curable only if removed early and completely.

The benign and some of the malignant papillomas respond fairly well to electrocoagulation, but, in the sessile infiltrating types of cancer of the bladder, such treatment has been disappointing and radiotherapy offers little more than palliation at best.

Since more than three-quarters of cancers of the bladder arise on its base, thereby involving the ureteral orifices or vesical sphincter,[24] excision of the tumor or partial cystectomy is usually not feasible. In the majority of cases, then, only total extirpation of the bladder offers a chance for cure.

We have chosen to divide this paper into two parts: I. A critical review of the literature on total cystectomy for cancer and, II, an analysis of personal cases of ablation of the bladder for malignant disease, with a brief description of our technic for prostato-seminal-vesiculo-cystectomy.

I. REVIEW OF THE LITERATURE

A. *History of Cystectomy.*—The first work on cystectomy was carried out on animals, and Gluck and Zeller[46] in 1881 reported the first successful cystectomies in dogs whose ureters were transplanted to the skin.

In 1887 Bardenheuer[5] performed the first total cystectomy on a man suffering from cancer of the bladder. The ureters were abandoned in the pelvis. The patient died of uremia fourteen days after the operation.

In 1889 Pawlik[107] carried out the first successful cystectomy, which was upon a woman. First, the ureters were transplanted to the vagina and, twenty-four days later, the bladder was extirpated. The patient was living sixteen years later. Kümmell[77] performed cystectomy with ureterourethral transplant in 1890. The patient died of shock. From a man suffering with cancer of the prostate with secondary invasion of the bladder, Küster,[78] in 1891, removed the prostate and bladder and

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was the first to combine cystectomy with ureteral transplantation to the rectum. The patient died, however, five days later, of peritonitis and pyelonephritis.

In this country, Martin[90] in 1899 was the first surgeon to remove the bladder for cancer. He performed a hysterocystectomy for malignancy of the uterus and bladder. The ureters were transplanted to the rectum. The patient died of peritonitis four days after operation.

Two hundred and thirty-six cases of total extirpation of the bladder for cancer have been tabulated from the literature and eighteen personal cases not previously reported have been added (Table I). This

TABLE I

TOTAL CYSTECTOMY

NUMBER OF CASES ---------- 254

OPERATIVE MORTALITY ---------- 87

PERCENTAGE MORTALITY ---------- 34.2%

METHOD OF DISPOSAL OF URETERS	ONE STAGE			TWO STAGES			TOTAL NO. OF CASES	PERCENTAGE MORTALITY
	NO. OF CASES	NO. OF POSTOPERATIVE DEATHS	PERCENTAGE MORTALITY	NO. OF CASES	NO. OF POSTOPERATIVE DEATHS	PERCENTAGE MORTALITY		
Abandoned in wound	11	5	45.4	--	--	--	11	45.4
Skin of abdominal wal	28	6	21.4	11	--	0.0	39	15.3
Lumbar ureterostomy	6	2	33.3	12	--	0.0	18	11.1
Nephrostomy	1	1	100.0	11	1	9.09	12	16.6
Urethra	6	6	100.0	--	--	--	6	100.0
Vagina	12	4	33.3	2	1	50.0	14	35.7
Bowel exclusion	11	6	54.5	6	2	33.3	17	47.0
Colon	63	38	60.3	74	15	20.3	137	38.0
Totals	138	68	49.2	116	19	16.2	254	34.2

latter group will be more fully discussed. The cases comprising the series recently reported by Higgins,[57, 58] Nesbit,[100] Kidd,[73] and others and some of the cases of Beer[8] and Smith[132] are not included in this compilation because details necessary for their evaluation were not recorded. Cystectomy for exstrophic bladders is not considered in this study.

In the past, Bovée (1901),[11] Druebert (1903),[30] Rafin (1905),[115] Verhoogen (1909),[145] Watson (1913),[152] Scheele (1923),[121] Papin (1925),[105] and Bompart (1931),[10] among others, have similarly listed the reported cases and discussed the subject.

B. *Indications for Total Cystectomy.*—The outstanding indication for complete cystectomy is for infiltrating cancer of the base of the bladder involving either the vesical outlet, ureteral orifices, or both. Other reasons for vesical ablation include recurring multiple papillomas of the

bladder, very large tumors of the bladder, and carcinoma of the prostate invading the neck of the bladder. Cystectomy has been carried out for other maladies as well, as may be seen in Table II.

TABLE II

TOTAL CYSTECTOMY

INDICATIONS

INDICATION	NUMBER OF CASES
Cancer of bladder	205
Squamous cancer of bladder	7
Cancer of bladder—uterus	8
Multiple papillomas of bladder	8
Cancer of prostate	8
Cancer of uterus	6
Sarcoma of bladder	5
Cancer of urethra	3
Cancer of cervix	2
Sarcoma of uterus	1
Cancer of vagina	1
Totals	254

With a few exceptions, cystectomy has been considered an operation of despair, which, to a large extent, explains the mortality attendant on this procedure. As has been noted, the majority of vesical malignancies take origin from the base of the bladder and invade the ureteral orifices, with resulting hydronephrosis and infection. These patients, from the renal standpoint, become poor surgical risks and their dilated thin-walled ureters are difficult to transplant, particularly to the colon.[73, 92] The mortality of cystectomy, however, has gradually fallen, due, in particular, to improvements in ureterointestinal anastomosis and to the employment of multistage procedures. An important step further in this direction will be made when early radical intervention is undertaken.[10, 36, 113] Quinby, for example, will perform cystectomy only if there are no evident metastases, if the patient has one normal kidney, and if his physical condition is sound.

C. *Methods of Diversion of Urine.*—1. *Ureters Abandoned in the Depths of the Wound:* When Bardenheuer performed the first cystectomy in man, he was forced to abandon the ureters in the pelvis because of the condition of his patient at the end of the operation. Verhoogen and de Graeuwe,[145] in 1896, deliberately followed this procedure. Eleven cases in which the ureters were treated in this manner were collected.

2. *Ureters Brought to the Skin of the Abdominal Wall:* This group includes ureteral transplantation to the hypogastrium, to the iliac region, and into the edges of the cystectomy wound.[16, 105]

Gigon[132] in 1856 and Agnew[105] in 1881 proposed ureterocutaneous transplant but did not use it clinically. In 1881 Gluck and Zeller successfully performed cystectomy in dogs and transplanted the ureters to the skin.

Laurenze[105] in 1888 brought a ureter, which he had accidentally cut, to the skin. The following year Le Dentu[105] chose this procedure for a woman with a uterine cancer which had invaded the ureters. Gynecologists often have had recourse to it when confronted with surgical accidents to the ureter. Wassiljew[150] in 1895, however, was the first to employ ureterocutaneous transplant with cystectomy. In all, thirty-nine cases utilizing this method have been recorded. Smith[132] performed the operation with cystectomy eleven times, but the cases were not published in detail and consequently are not included in this study.

Suturing the ureters to the edges of the cystectomy wound naturally presupposes a one-stage procedure; further, as the wound heals, scar tissue causes stenosis of the orifices. Chute[19] and Fuchs[41] in 1925 were the last to utilize this method.

Papin[105] stressed the superiority of iliac ureterostomy because of the low mortality associated with it, the efficiency of the collecting apparatus, and the accessibility of the ureters for dilatation and lavage.

Maximovitsch[92] and Smith,[132] among others, noted the frequent occurrence of sloughing of the distal ends of the ureters, often necessitating reoperation. Many surgeons object strenuously to the apparatus employed for collection of the urine.

3. *Lumbar Ureterostomy:* Rovsing,[119] in 1906, was the first surgeon to perform cystectomy with lumbar ureterostomy and Whiteside,[43] in this country, carried out a similar procedure in the same year. Eighteen such cases have been collected. The disadvantages of this type of ureterostomy, in addition to those held in common with all cutaneous transplants, are the inaccessibility of the ureters to the patient who must care for them and the unsatisfactory collecting apparatus necessary because of their dorsal position.

4. *Nephrostomy:* Nephrostomy has long been employed as a means of temporary or permanent drainage of the kidney,[16] but it was not until 1905 that Watson[151] first suggested its use in conjunction with cystectomy. Three years later, Marion[89] performed the first nephrostomy-cystectomy, which he accomplished in two stages. Cabot,[15] in 1909, followed a similar procedure in a man with cancer of the prostate, cystectomy and partial prostatectomy being done. Twelve such cases were found.

Nephrostomy has the advantage, in common with ureterocutaneous transplants, of a low rate of mortality, but the operation of nephrostomy causes damage to the kidney, the drainage of the lower half of the kidney is poor, and the dorsal position of the collecting apparatus is inconvenient.

The advocates of nephrostomy and ureterocutaneous transplant point with pride to the low operative mortality associated with these pro-

cedures. They also prefer having the ureters accessible for dilatations and lavage and claim that the special apparatus for collecting the urine is efficient.

Many surgeons, including Papin,[105] Fey[36] and Rovsing,[119] believe that cutaneous transplant (anterior or lumbar) causes less damage to the kidneys than any other method of urinary diversion.

5. *Ureters Transplanted to the Urethra:* Sonnenburg[2] in 1882 was the first surgeon to employ transplant to the urethra. This was in a case of exstrophy. In 1890, Kümmell[77] performed urethral transplant in conjunction with cystectomy. He brought the right ureteral catheter out through the urethra. In all, six cases of transplant to the urethra have been reported, with six deaths. Obviously, such a transplant presupposes a one-stage operation, which is a disadvantage. Certainly the results in these cases suggest why the procedure has been abandoned.

6. *Ureters Transplanted to the Vagina:* Pawlik,[107] in 1889, not only performed the first successful cystectomy but was the first to utilize ureterovaginal transplant. At the first stage he carried out the transplant vaginally, using indwelling ureteral catheters. Twenty-four days later extraperitoneal vaginal cystectomy was done for cancer. The patient was alive and well sixteen years after operation.

In all, fourteen such cases have been tabulated. An attempt is usually made to obtain continence by closing the vaginal orifice, the new bladder, the vaginal cavity, emptying through the urethra. This, however, has not been particularly successful in practice so that usually collecting devices are necessary and the ureters are open to the possibility of dilatation and ascending infection. The ureters cannot be reached, as they can in ureterocutaneous transplants, for dilatations and lavages. This method of dealing with the ureters, therefore, has fallen into disrepute and has not been used during the past twenty years.

7. *Ureters Transplanted to Excluded Bowel:* The ureters may be transplanted to a segment of either partially or totally excluded bowel.

Mauclaire[145] in 1894 first suggested total exclusion of the rectosigmoid by division of the sigmoid loop, sigmoid colostomy, and closure of the proximal end of the distal limb, with implantation of the ureters into the latter structure. This he performed in dogs.

Krönig[76] first employed this method in conjunction with cystectomy in 1906. The first stage consisted of exclusion of the rectosigmoid and sigmoid colostomy. Twelve days later, the ureters were transplanted into the blind sigmoid and hysterocystectomy for sarcoma of the uterus was carried out. The patient was alive five years later. A similar procedure was carried out by Thies,[137] Schmieden,[123] and Lindström,[83] the latter two surgeons transplanting the ureters at a separate operation before cystectomy was done.

In 1912, Lemoine[80] performed a cystectomy and transplanted the ureters to the rectum. Later, because the patient suffered from renal

infection, he excluded the rectum, making it communicate with the urethra, and attempted to pull the sigmoid through the anal sphincter to allow both fecal and urinary continence. Unfortunately, the sigmoid retracted and the patient died of sepsis.

Verhoogen[145] in 1908 and Lengemann[81] in 1911 utilized a different type of procedure. Verhoogen excluded the ileocecal region while Lengemann excluded the entire ascending colon; both carried out iliotransverse colostomy, appendicocutaneous transplant, and anastomosis of the ureters to the cecum.

Berg[60] isolated a segment of ilium, anastomosed one end to the sigmoid and transplanted the ureters to the blind end, thereby forming a partially excluded loop of bowel into which the urine drained.

In all, seventeen cases based on the foregoing principle were found.

These procedures, complicated as they are, were proposed because of the desire to abolish ascending renal infection. Judging by the cases reported, however, the advantages are only theoretical. Pyelonephritis is not eliminated, stenosis of the ureteral orifices occurs just as readily as in ureterointestinal anastomosis, and the amount of surgery is greatly increased. In the face of the recent successes experienced with ureteroenterostomy, exclusion of the bowel has become unnecessary.

8. *Ureterointestinal Anastomosis:* Gluck and Zeller,[46] in 1881, made an unsuccessful attempt to combine cystectomy and ureterointestinal anastomosis in dogs.

Ten years later, Küster[78] performed rectoureteral transplant and a perineoabdominal extraperitoneal prostatocystectomy, but without success. Tuffier,[141] in 1895, was the first to perform an effective transplant of the ureter to the bowel. Martin,[90] in 1899, was the first surgeon in this country to attempt the operation in conjunction with cystectomy. One hundred and thirty-seven cases of cystectomy have been collected in which the ureters were transplanted to the colon. The subject of ureterointestinal implantation, with a critical study of the surgical principles involved, has been recently made and need not be repeated here.[60]

Many surgeons, particularly the French, have objected strenuously to intestinal transplant because of its higher mortality from operation, arising from peritonitis and pyelonephritis, and later morbidity from progressive hydronephrosis and renal infection. Bompart[10] even stated that "transplantation of the ureters to the intestine should be completely abandoned in total cystectomy for cancer." Beer,[8] though he feels that, theoretically, a procedure allowing sphincteral control of the urine is to be preferred, for practical reasons resorts to ureterocutaneous transplant with which he enjoys a low operative mortality and fairly satisfactory results.

Many of the Russian, German, British, and American surgeons, however, have persisted in developing ureteroenterostomy, with the result that, of the 75 cases of cystectomy with ureterointestinal anastomosis

published since 1926, only 19 patients, or 25.3 per cent, died from the operation. By 1927 Fedoroff[92] had done 9 cases without a death. Coffey,[22] in 1933, reported 11 cases with only 3 deaths, and two years later Quinby[112] listed 10 cases with only 2 deaths. Among our last 14 patients suffering from cancer, there were 3 deaths following ureteroenterostomy and radical removal of the bladder and adjacent organs. As may be noted from Table I, the mortality for ureterointestinal transplant done in two stages was only 20.3 per cent. This figure, however, does not take into account the patients who died of the operation for transplanting the ureters, thereby not entering into these computations. This point will be discussed later. One factor which has decreased the mortality in this group has been the employment of a two- or three-stage procedure. The mortality will fall further when the patients are operated upon early, when the cancer is limited to the bladder, and when the kidneys are healthy and the ureters are not dilated.

Transplantation of the ureters to the bowel has the advantage of having the urine controlled by a sphincter. This factor, however, must be weighed against the lower mortality associated with cutaneous transplants.

D. *The Technique of Cystectomy.*—1. *In the Male:* If the cancer involves only the lateral or posterior wall of the bladder without affecting the prostate or seminal vesicles, cystectomy may be all that is necessary, although it has been proved that the limits of a malignant growth cannot be ascertained by inspection. Microscopically, isolated islands of cancer cells may be found far removed from the primary growth. In most instances, therefore, complete prostatoseminal-vesiculocystectomy is indicated; it becomes necessary when the prostate is the site of primary cancer or is secondarily invaded by tumor.

Since a cancerous bladder is always septic, it should be removed unopened, although some surgeons insist upon exploratory cystotomy before cystectomy is attempted. Because of the potential infection, most surgeons prefer an extraperitoneal technique. In many instances, however, the serosa covering the fundus is either adherent or is involved by cancer. In such cases the serosa must be resected and left on the bladder, whereupon the peritoneum is repaired.

Most operations have been performed through a suprapubic approach, although a combined abdominoperineal or perineoabdominal technique has many advantages.

If the suprapubic approach is utilized,[2] the serosa may be separated from the bladder laterally and posteriorly, the bladder stripped anteriorly from the symphysis by cutting the pubovesical ligaments, the lateral vesical ligaments divided, thereby controlling the major blood supply, and the ureters sectioned. The dissection is than carried posteriorly, separating the bladder from the rectum and dividing the urethra between the prostate and bladder.

If the prostate and seminal vesicles as well are to be removed suprapubically, they are separated from the rectum posteriorly, the prostate is freed all around, and the distal portion of the prostatic urethra is sectioned.[37]

Harris,[53] in 1901, performed a similar operation except that he first carried his dissection anteriorly, cut the pubovesical ligaments, and freed the anterior surface of the prostate. He then divided the membranous urethra and, by a retrograde method, pulled the prostate, vesicles, and bladder out from below upward. Coffey,[22] Ward,[149] and one of us (F. H.)[59] have utilized this technique more recently. We, however, have abandoned it because of the occurrence of bleeding from the plexus of Santorini before exposure is sufficient to permit its control. Beer[8] criticized this technique because, in his opinion, it tends to milk cancer cells into the lymphatics.

In 1891, Küster[78] first utilized a combined perineoabdominal approach. It has also been well described by Albarran.[2] With the patient in the lithotomy position, the prostate, seminal vesicles, and base of the bladder are separated from surrounding tissues by a technique similar to that of Young's radical perineal prostatectomy. The prostatic urethra is divided. A suprapubic incision is then made and the bladder freed, allowing removal of the organs *en masse*.

If the prostate is involved by cancer, it is more easily separated by the perineal route; whereas, if it is free of malignancy, it is more easily stripped off the rectum from above.

We prefer an abdominoperineal technique which is described in the second part of this paper.

Beer[6] performs suprapubic extirpation of the bladder with transection of the prostate with the cautery. Ward[149] has also used this method.

Drainage of the resultant extraperitoneal suprapubic and pelvic space is essential. Many surgeons prefer a Mikulicz or quarantine pack, while others use tube drains. Perineal drainage is a great advantage because of its dependency. Poor drainage of this dead space, which fills with serum, may lead to the formation of an abscess.

2. *In the Female:* The technique most often used in the female is suprapubic extirpation.[2, 79, 136] If the uterus or its appendages are involved, cystohysterectomy may be indicated.[3]

If the cancer involves the urethra, or is primary there, urethrocystectomy is necessary.[33, 40] This may be accomplished by a vaginoabdominal or abdominovaginal route. One of us (F. H.) performed cystectomy solely by the vaginal route in one case. Recently, Shaw[127] evolved a ureterorectal transplant and cystectomy by the vaginal route. This method has not yet been employed clinically.

E. *Number of Stages.*—Most surgeons are now convinced that multiple-stage operations are of distinct advantage in these cases.[34, 36, 121] Ureteral transplantation, particularly if to the colon, is enough of a tax

on a patient without adding the shock of cystectomy. There are many plans for two- or more-stage procedures. Both ureters may be transplanted at one stage, or one ureter may be transplanted at the time of cystectomy. A three-stage procedure is also utilized; i.e., two transplants and cystectomy.

Table I shows the mortality depending on the number of stages used. This comparison, however, is not a true picture of results because, particularly in cases of ureterointestinal anastomosis, many patients for whom cystectomy is planned die from the anastomosis and therefore the cases are not recorded. Obviously, cystectomy itself has a low mortality, probably under 5 per cent, and even some of these deaths are due in reality to factors secondary to the transplanted ureters.

F. *Results of Cystectomy.*—In the 254 cases of total cystectomy, there were 87 operative deaths, a mortality of 34.2 per cent (Table III). This is the lowest rate yet reported for cystectomy and is due to the inclusion of recent cases in which the results have been greatly improved. The actual mortality at the present time, therefore, is lower than the figure given.

Among those 11 patients in whom the ureters were abandoned in the depths of the wound, there were 5 operative deaths. None was reported as living more than two years. Because of its high mortality and poor results, this method has become of historical interest only.

TABLE III

SUMMARY OF RESULTS

METHOD OF DISPOSAL OF URETERS	TOTAL NO. OF CASES	NO. OF POST-OPERATIVE DEATHS	SURVIVAL BUT WITHOUT FOLLOW-UP	SURVIVAL (DEAD OR ALIVE AT LAST REPORT)			
				UNDER 1 YR.	1 TO 5 YR.	5 TO 10 YR.	10 YR. OR MORE
Abandoned in wound	11	5	1	3	2	--	--
Skin of abdominal wall	39	6	4	19	8	2	--
Lumbar ureterostomy	18	2	7	7	1	0	1
Nephrostomy	12	2	--	5	3	0	2
Urethra	6	6	--	--	--	--	--
Vagina	14	5	1	6	1	0	1
Bowel exclusion	17	8	1	3	3	2	--
Colon (One stage)	63	38	1	12	10	1	1
Colon (Two stages)	74	15	9	27	19	4	--
Totals	254	87	24	82	47	9	5

Among those 39 patients in whom the ureters were transplanted to the skin of the abdominal wall, there were only 6 deaths from the operation, or 15.3 per cent, in spite of the fact that the majority of the operations were performed in one stage. This figure compares favorably with resections of the bowel for cancer and is the major reason why many surgeons utilize it.

Only about 1 of 4 patients comprising this group lived one year or more, though Beer reported 2 patients alive after eight and one-half and

four years respectively and 1 who died of cancer and tuberculosis five years after operation. It is interesting to note, however, that in the 11 cases in which operation was done in two stages, there were no operative deaths.

Among those 18 patients for whom lumbar ureterostomy was performed, there were 2 operative deaths, or 11.1 per cent. In only 1 case was a good result obtained; Brongersma's patient was alive fifteen years after operation when last reported. Only 1 other patient lived more than one year. In this group, also, it may be seen that no operative deaths resulted among those patients for whom a two-stage operation was chosen.

There were 2 operative deaths in the nephrostomy group of 12 cases but in 1 of these (personal case) prostatoseminal-vesiculocystectomy was carried out only because of severe sepsis of the bladder from which the patient would have shortly succumbed. He was not operable. Only this one patient, of 11 treated by a two-stage procedure, died from the operation.

One of Marion's patients was still living thirteen years after the operation and one of Thomas' patients died sixteen years after operation of pulmonary and intestinal tuberculosis. Smith and Marion each reported a case in which death occurred three years after operation, from carcinomatosis.

As noted above, under the discussion of the six patients subjected to cystectomy in addition to transplantation of the ureters to the urethra, all died from the operation.

In the group of 14 cases in which vaginoureteral transplant was done, there were 5 operative deaths, or 35.7 per cent. Except for Pawlik's patient, who was living sixteen years after operation, the results have been poor, only 1 other patient having lived more than one year.

The ureters were transplanted to partially or completely excluded bowel in 17 cases, with 8 postoperative deaths, or 47 per cent. Krönig's patient was still alive five years after operation and 1 treated by Thies was living three years after operation. Gironcoli reported on a patient who died of uremia after five and one-half years, while Thies's other patient died four and one-half years after operation of cachexia and pulmonary tuberculosis.

Ureterointestinal transplant was carried out in conjunction with cystectomy in 137 cases, with 53 deaths, a mortality of 38 per cent. This figure, of course, does not take into account those patients who died as a result of preliminary diversion of the urinary stream. It is not fair to quote the mortality for the whole group and accept it as the true mortality at the present time because recent improvements in ureteroenterostomy have lowered the operative risk. Thus, in the 75 cases reported since 1926, there were only 19 operative deaths, a mortality of 25.3 per cent.

The following authors had patients who, when last reported, had been living three years or more: Fedoroff, six years; Coffey, four years; Hinman, three and one-half years; Coffey, three years; Ssolowoff, three years.

Those listed below reported cases in which their patients lived four years or more but died from the causes indicated:

TABLE IV

AUTHOR	YEARS OF LIFE AFTER OPERATION	CAUSE OF DEATH
Fishel	13	Bronchopneumonia, no recurrence
Vehrs	6	Recurrence of cancer
Von Winiwarter	5½	Pyelonephritis, pulmonary abscess
Ward	5½	Carcinomatosis
Fedoroff	5	Recurrence of cancer
Hinman	4	Recurrence of cancer

Of the 73 patients who survived operation, on whom complete or partial follow-up records were secured, 35 lived one year or more.

The chief causes of operative death following cystectomy were acute pyelonephritis and shock. Recurrence or metastasis of cancer caused the majority of late deaths. Other causes have been listed in Table V.

G. *Discussion.*—In order to arrive at a more accurate conclusion concerning the risk of total extirpation of the bladder to the patient of today, we must base our study on cases in which the urinary stream was diverted by abdominal and lumbar ureterocutaneous transplant and ureteroenterostomy. To evaluate the latter method, we should consider only recent cases because of improved surgical principles now employed.

From Table I, it may be concluded that cystectomy combined with ureterocutaneous transplant either to the abdominal wall or the lumbar region promises a surgical mortality of about 15 per cent.

If a two-stage ureteroenterostomy-cystectomy is performed and if the patient survives the primary ureteral transplant, the mortality will be about 20 per cent. If those dying from the first stage of the procedure are included, the mortality will be above 30 per cent. The average duration of life of those patients surviving the operation is about the same, whether their ureters are transplanted to the skin (anteriorly or posteriorly) or to the intestine. The surgeon must decide which is of most importance to the patient, the risk of the operation or the patient's comfort after the operation.

The hope for improvement in the results of cystectomy lies in early radical surgery when cancer is limited to the bladder, when the ureters are healthy and the condition of the patient is such that he can stand an extensive surgical procedure. This is particularly true in ureteroenterostomy.

Lack of space does not allow the tabulation of the published cases gathered from the literature. On pages 864 and 865 (Table VI), are listed the date of cystectomy, the name of the surgeon (or reporter), and the references. The cases are divided according to the method of disposal of the ureters.

TABLE V

DISPOSAL OF URETERS	TOTAL NUMBER OF CASES	NO. OF POSTOPERATIVE DEATHS	CAUSE OF POSTOPERATIVE DEATH															CAUSE OF LATE DEATH																
			PYELONEPHRITIS	SHOCK	ANURIA	UREMIA	PERITONITIS	PNEUMONIA	PYELONEPHRITIS, PERITONITIS	PYELONEPHRITIS, WOUND ABSCESS	PULMONARY EDEMA	SEPSIS (NONRENAL)	WOUND ABSCESS, SEPTICEMIA	MASSIVE COLLAPSE OF LUNG	PYONEPHROSIS	CAUSE NOT NOTED	RECURRENCE OF CANCER	PYELONEPHRITIS	CARCINOMATOSIS	PYONEPHROSIS	PULMONARY TUBERCULOSIS	NECK INFECTION—SEPTICEMIA	HYDRONEPHROSIS	CORONARY OCCLUSION	MELENA	UREMIA	HEMORRHAGE FROM KIDNEY	SEPSIS	NEPHRITIS	BOWEL OBSTRUCTION	CAUSE NOT NOTED
Abandoned in wound	11	5	1	1		1	1									1	1	2														
Abdominal wall	39	6	1												1	4	6	1	3	1	1	1	1	1				1				
Lumbar ureterostomy	18	2		1		1											2	1							1							1
Nephrostomy	12	2		1												1			3	1	1						1					1
Urethra	6	6		3		1										2													1			
Vagina	14	5		1		1		1								2		2														
Bowel exclusion	17	8	3	1	1	1										2	1			2	1											1
Colon	137	53	16	13	2		3	3	3	2	1	2	1	1		6	16	6	5			1				1	1					11
Totals	254	87	21	21	3	5	4	4	3	2	1	2	1	1	1	18	26	12	11	4	3	2	1	1	1	1	1	1	1	1	1	14

II. SUMMARY OF PERSONAL EXPERIENCE WITH TOTAL CYSTECTOMY FOR CANCER

A. *Analysis of Personal Cases.*—There has been nothing published in the literature dealing with the true risk to the patient for whom ureteroenterostomy and total cystectomy are planned. All the authors who have presented statistics regarding their personal results with cystectomy have failed to include the patients for whom radical surgery was originally intended but who for one reason or another had preliminary ureteral transplantation only. Since 1925 we have performed ureteroenterostomy on fifty patients suffering from cancer.

1. *Ureteroenterostomy Without Cystectomy:* Twenty-four of these patients never came to radical removal of their tumors (Table VII). Eighteen of this group died as a result of the ureteroenterostomy and 5 patients died within ten months of surgery, while the other patient committed suicide one year and three months later; he was inoperable.

We have classified these patients into two groups, operable and inoperable, based on clinical findings before surgery and at the operating table. In this group of 24 patients who had only preliminary diversion of the urinary stream, only 7 had cancers which might have proved operable. Six of the 7 died from the effects of the diversion, 1 of shock, 4 of renal sepsis, and 1 of circulatory failure. The other patient died ten months later of renal infection.

Of the remaining 17 patients, 12 died after operation: 5 of renal sepsis, 3 of generalized peritonitis due to faulty surgical technique, 2 of bronchopneumonia, 1 of bilateral hydronephrosis, and 1 of lobar pneumonia. Of the other 5 patients, 4 died of cancer within ten months, while 1 committed suicide one year and three months after surgery.

2. *Nephrostomy With Cystectomy:* Nephrostomy and nephrectomy for bilateral hydronephrosis were performed on a man for whom ureteroenterostomy and finally cystectomy were planned. Because of severe sepsis of the bladder, however, abdominoperineal prostatoseminal-vesiculocystectomy was carried out twenty-five days later. Not only was he a very poor risk from the standpoint of sepsis and renal impairment, but he was found to be inoperable. He died twenty-four hours after the second operation.

3. *Ureteroenterostomy With Cystectomy:* Twenty-five of the 50 patients with cancer underwent ureteroenterostomy and cystectomy, all during the past five and one-half years (see tabulated personal cases appended). Ten of these (40 per cent) died following operation, while 6 are living and well up to three and one-half years after surgery.

Of these 25 patients, 17 were considered operable. Seven (41.1 per cent) of these died shortly after cystectomy; 2 of generalized peritonitis, 2 of pelvic abscess and sepsis, 1 of renal sepsis, 1 of pneumonia, and 1 of massive pulmonary atelectasis. Three patients died later of cancer,

TABLE VI

DATE	SURGEON	DATE	SURGEON	DATE	SURGEON
	Cystectomy With Ureters Abandoned in Wound				
1887	Bardenheuer[5]	1903	McCosh[93, 125]	1913	Rafin[152]
1896	Verhoogen[145]	1903	McCosh[93, 125]	1920	Egger[31]
1898	Bardenheuer[145]	1905	Hamonic[52]	1920	Egger[31]
1898	Bardenheuer[145]			1926	Chute[19]
	Cystectomy With Ureters to Skin of Abdominal Wall				
1895	Wassiljew[150]	1913	Keyes[43]	1927	Hazanoff[55]
1901	Harris[53]	1915	Cifuentes[20]	1927	Hazanoff[55]
1902	Garre[49]	1915	Beer[6]	1927	Hazanoff[55]
1905	Verhoogen[145]	1915	Beer[6]	1927	Beer[6]
1905	Bruni[10]	1919	Beer[6]	1927	Hazanoff[55]
1908	Marion and Legueu[89]	1919	Nassauer[121]	1928	Hazanoff[55]
		1920	Papin[105]	1928	Maister[86]
1910	Héresco[56]	1922	Messtorff[95]	1928	Beer[6]
1910	Zuckerkandl[39]	1922	Rochet and Thévenot[118]	1928	Beer[6]
1910	Tenney[43]			1930	Just[71]
1911	Héresco[56]	1925	Fuchs[41]	1931	Godard[47]
1911	Héresco[56]	1925	Fuchs[41]	1932	Winsbury-White[156]
1911	Keller[43]	1925	Beer[6]		
1912	Héresco[56]	1925	Chute[19]	1935	Walters[148]
	Cystectomy With Lumbar Ureterostomy				
1906	Rovsing[119]	1908	Straus[135]	1915	Crosbie[26]
1906	Rovsing[119]	1909	Martin[91]	1925	Fuchs[41]
1906	Whiteside[43]	1911	Brongersma[13, 14]	1927	Antonucci[3]
1907	Rovsing[119]	1912	Furniss[42]	1930	Hryntschak[68]
1907	Fenwick[35]	1913	Marion[103]	1934	Freiberg[40]
1908	Fenwick[35]	1913	Morton[99]	1935	Freiberg[40]
	Cystectomy With Nephrostomy				
1908	Marion[118]	1909	Marion[118]	1917	Smith, G. G.[129]
1909	Cabot[15]	1909	Marion[118]	1922	Joseph[70]
1909	Heitz-Boyer[28]	1912	Marion[103]	1934	Hyman[66]
1909	Marion[118]	1914	Thomas[138]	1937	Hinman (see text)
	Cystectomy With Transplant to Urethra				
1890	Kümmell[31, 77]	1896	Albarran[1]	1903	Keyser[151]
1895	Lindner[82]	1897	Goepel[48]	1920	Egger[31]
	Cystectomy With Transplant to Vagina				
1889	Pawlik[107]	1900	Mann[88]	1908	Pollosson[136]
1894	Kossinsky[96]	1901	Mann[88]	1910	Cuneo[136]
1896	Zeller[158]	1901	Smith, A. L.[145]	1910	Pollosson[136]
1897	Modlinski[98]	1902	Robson[117]	1925	Fuchs[41]
1898	Modlinski[98]	1908	Seidel[126]		
	Cystectomy With Ureters to Excluded Bowel				
1906	Krönig[76]	1912	Lemoine[80]	1922	Scheele-Schmieden[121]
1907	Berg[152]	1912	Thies[137]		
1908	Verhoogen[145]	1913	Thies[137]	1926	Lindström[83]
1908	Verhoogen[145]	1921	Schmieden[10]	1929	Gironcoli[45]
1908	Berg[152]	1921	Rubritius[121]	1934	Gironcoli[45]
1911	Lengemann[81]	1921	Schmieden[122, 123]	1937	Gironcoli[45]
	Cystectomy With Ureters Transplanted to Colon				
1891	Küster[78]	1920	Egger[31]	1931	Ward[149]
1895	Tuffier[140]	1921	Loewe[84]	1931	Ssolowoff[134]
1895	Giordano[145]	1921	Fedoroff[31]	1931	Coffey[22]
1896	Chalot[11]	1922	Smith, G. G.[132]	1931	Smith, G. G.[132]
1897	Turetta[142]	1922	Smith, G. G.[132]	1931	Ssolowoff[134]
1897	Giordano[145]	1923	Lindström[83]	1931	Ssolowoff[134]
1897	Wendel[154]	1923	Fishel[38]	1931	Coffey[22]
1897	Küster[78]	1923	Fedoroff[34]	1931	Coffey[22]

TABLE VI—CONT'D

DATE	SURGEON	DATE	SURGEON	DATE	SURGEON
1898	von Winiwarter[65]	1924	Smitten[133]	1931	Riches[116]
1898	Schede[151]	1925	Pleschner[109]	1931	Riches[116]
1899	Weljaminow[136]	1925	Smith, G. G.[132]	1932	Bell[9]
1899	Krause[74]	1925	Ward[149]	1932	Ward[149]
1899	Martin[90]	1925	Chute[19]	1932	Dodd[29]
1899	Martin[90]	1925	Fedoroff[34]	1933	Ward[149]
1900	Whiteside[43]	1925	Simon[128]	1933	Hinman*
1901	Krause[75]	1925	Simon[128]	1933	Hinman[60]
1902	Woolsey[157]	1926	Maximovitsch[92]	1933	Hinman[61]
1902	Lund[85]	1926	Quinby[111]	1934	Hinman[59]
1903	Vaughan[143]	1926	Smith, G. G.[130, 132]	1934	Hinman[61]
1903	Schwyser[125]	1926	Fedoroff[34]	1934	Smith, G. G.[132]
1904	Carson[17]	1927	Maguire[87]	1934	Smith, G. G.[132]
1904	Wilms[155]	1927	Smith, G. G.[132]	1934	Hinman[61]
1905	Lindner[136]	1927	Pleschner[110]	1934	Hinman[61]
1905	Quervain[152]	1928	Ssolowoff[134]	1934	Ward[149]
1906	Verhoogen[145]	1928	Coffey[22]	1934	Hinman[61]
1906	Schwartz[124]	1928	Ssolowoff[134]	1934	Hinman*
1906	Wilms-Jaeger[159]	1928	McIvor[94]	1935	Griffin[50]
1907	Pauchet[106]	1928	Ssolowoff[134]	1935	Hinman[63]
1907	Rouffart[145]	1929	Ssolowoff[134]	1936	Quinby[114]
1907	Rouffart[145]	1929	Smith, G. G.[132]	1936	Ward[149]
1907	Enderlen[32]	1929	Aschner[4]	1936	Hinman*
1907	Lauenstein[152]	1929	Coffey[22]	1936	Hinman*
1909	Petrow[108]	1929	Ssolowoff[134]	1936	Hinman*
1911	Fedoroff[33]	1930	Hryntschak[68]	1936	Hinman*
1911	Beer[6]	1930	Ssolowoff[134]	1937	Hinman*
1911	Hagner[51]	1930	Coffey[22]	1937	Hinman*
1912	Moscowicz[10]	1930	Coffey[22]	1937	Hinman*
1912	Samborsky[120]	1930	Coffey[22]	1937	Hinman*
1913	Gorache[120]	1930	Coffey[22]	1937	Hinman*
1915	Fedoroff[33]	1930	Ssolowoff[134]	1937	Hinman*
1917	Fedoroff[33]	1930	Coffey[22]	1937	Hinman*
1918	Smitten[133]	1930	Nitch[101]	1938	Hinman*
1918	Jefferson[69]	1931	Nitch[102]	1938	Hinman*
1918	Smith, G. G.[132]	1931	Coffey[22]	1938	Hinman*
1920	Egger[31]	1931	Pedroso[23]	1939	Hinman*
1920	Egger[31]	1931	Vehrs[144]		

*Previously unreported cases.

one in four years, one after one year and eight months, and one seven months after cystectomy. One patient died after two years and four months, autopsy revealing bilateral atrophic pyelonephritis with renal insufficiency, without evidence of cancer. The remaining 6 patients are living and well for the following periods: three and one-half years, two years and four months, two years and three months, one year, six months, and two months.

Eight patients in this group were found to be inoperable. Three died after operation, 1 of acute pyelonephritis, 1 of renal sepsis and pelvic abscess, and 1 of a pelvic abscess and pneumonia. The other 5 patients died later of cancer, 1 in two years and eight months, 1 after one year and six months, 1 at one year, and 2 at the end of four months.

Of these 50 cases, then, only 24 were considered to be operable and 3 of these patients died later of cancer. Therefore, in the light of our

TABLE VII

FIFTY PATIENTS WITH CARCINOMA
24 had ureteroenterostomy only:
7 operable
6 died following surgery
1 died later of renal infection
17 inoperable
12 died following surgery
4 died later of cancer
1 committed suicide
1 had nephrostomy and cystectomy:
Inoperable; died following surgery
25 had ureteroenterostomy and cystectomy:
17 operable
7 died following surgery
3 died later of cancer
1 died later of atrophic pyelonephritis
6 living and well
8 inoperable
3 died following surgery
5 died later of cancer

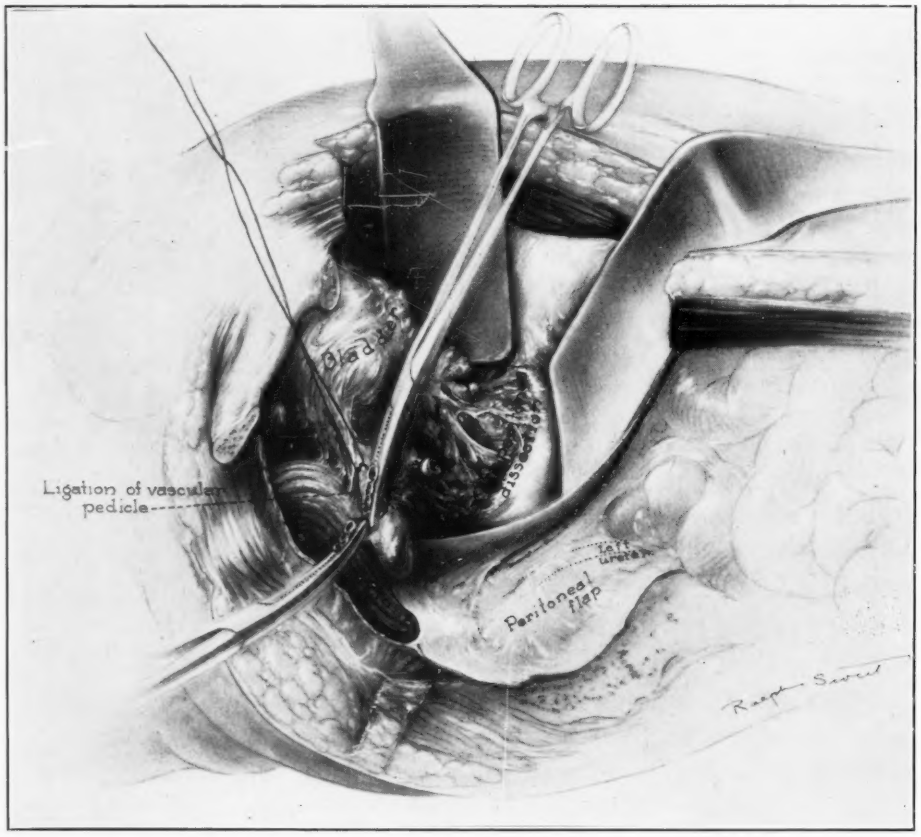

Fig. 1.—Method of prostatoseminal-vesiculocystectomy. Suprapubic extraperitoneal exposure of the bladder with ligation of the right and left vascular pedicles.

present knowledge, only 21 had a chance for cure. Thirteen of these died from the effects of surgery, 2 died later of renal infection, while 6 (28.5 per cent) are living and well.

A lower mortality would have resulted had candidates for such radical surgery been more carefully selected. Many of the earlier patients had ureteroenterostomy done by various methods (Higgins, Coffey I, Coffey II). Now that experimental and clinical observations have led to a standardized technique for transplantation of the ureters to the sigmoid and cystectomy or prostatoseminal-vesiculocystectomy, surgical errors have decreased and our results have improved correspondingly.

Approximately one-half the patients under discussion were known to be inoperable, but most of these operations were done in the earlier years. Of the 11 patients who underwent cystectomy during the past two and one-half years, only 1 was inoperable to our knowledge. Only 3 of the

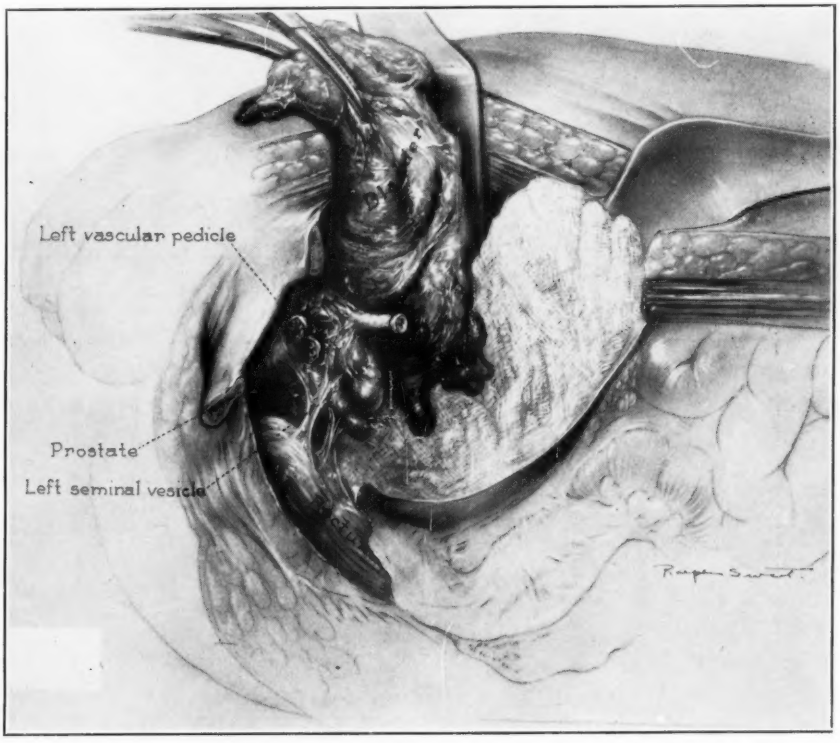

Fig. 2.—Method of prostatoseminal-vesiculocystectomy. By blunt dissection the bladder is freed down to the prostate anteriorly and the vesicular area posteriorly.

last 14 patients upon whom cystectomy was performed died in the hospital. In 1, a pelvic abscess developed when one ureter pulled out of the sigmoid; the second died of massive collapse of the lung; the third, at autopsy, had a perfect technical result and only bronchopneumonia was found to explain his death.

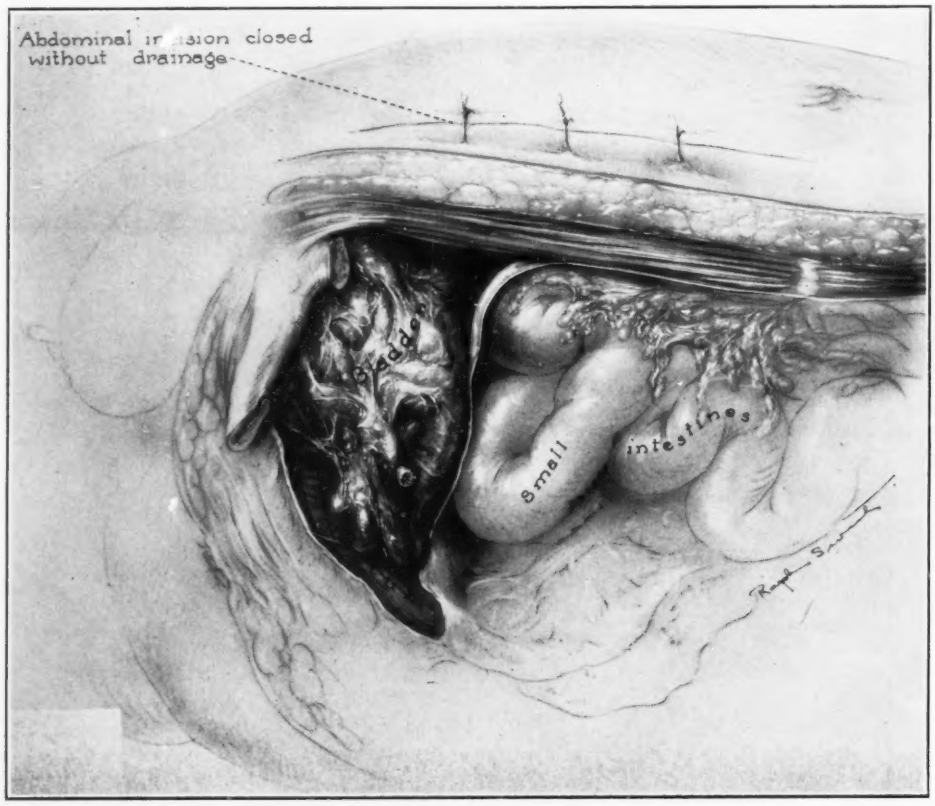

Fig. 3.—Method of prostatoseminal-vesiculocystectomy. The abdominal incision is closed without drainage and the patient placed in lithotomy position.

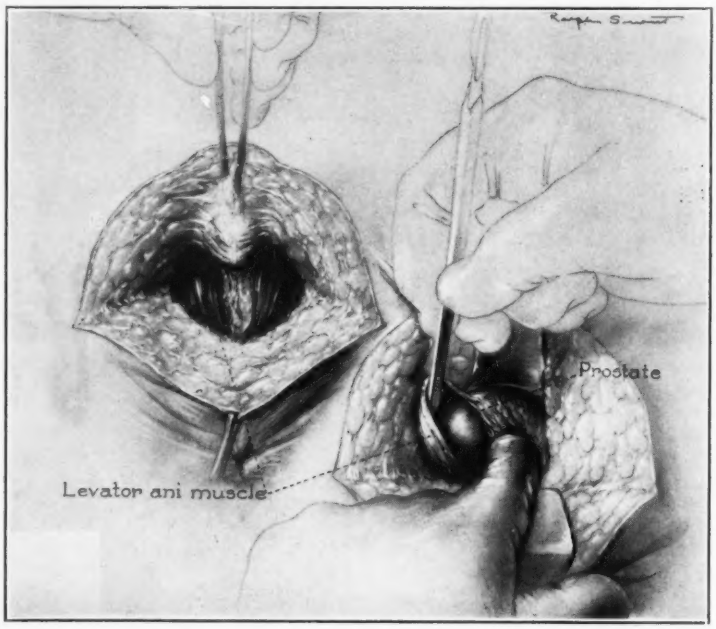

Fig. 4.—Method of prostatoseminal-vesiculocystectomy. Perineal exposure of the prostate and seminal vesicles.

III. TECHNIQUE OF ABDOMINOPERINEAL TOTAL PROSTATOSEMINAL-VESICULOCYSTECTOMY

Table VIII lists the various approaches utilized in this group of personal cases. In women, cystectomy only has been performed unless the urethra has been primarily or secondarily involved. We have always planned radical excision of the prostate and seminal vesicles along with the bladder in men, but in one case this was not possible because of the extent of cancer in the pelvis. Recently, a combined abdominoperineal

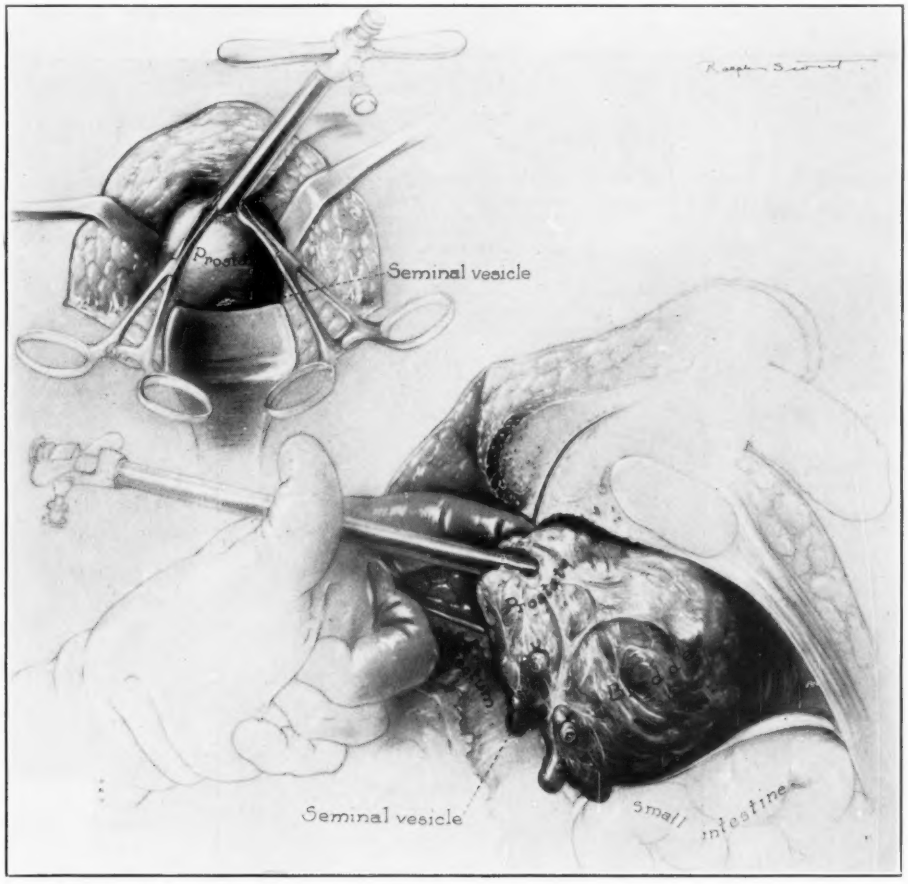

Fig. 5.—Method of prostatoseminal-vesiculocystectomy. The prostate and vesicles have been dissected free as for a radical prostatoseminal vesiculectomy.

operation has been evolved which seems to offer many advantages. It involves a clean excision of the bladder, prostate, and seminal vesicles, combining suprapubic cystolysis and radical perineal prostatoseminal-vesiculocystectomy.

The first operative stage consists usually of right ureteroenterostomy.[63] Two or three weeks later, the left ureter is transplanted to the sigmoid and radical removal of the bladder is carried out in the following manner:

After the ureter has been transplanted, dissection of the peritoneum from the fundus and posterior surface of the bladder is begun before the peritoneum of the wound proper is closed. After closure of the peritoneum, working extraperitoneally, the serosa is freed from the bladder well down into the cul-de-sac (Fig. 1). Next the bladder is freed an-

TABLE VIII

TECHNIQUE USED IN 26 RADICAL OPERATIONS FOR CANCER

18 men:	
Abdominal cystectomy	1
Abdominal prostatoseminal-vesiculocystectomy	11
Abdominoperineal prostatoseminal-vesiculocystectomy	6
8 women:	
Vaginal urethrocystectomy	1
Abdominal cystectomy	5
Vaginoabdominal urethrocystectomy	1
Abdominovaginal cystourethrectomy	1

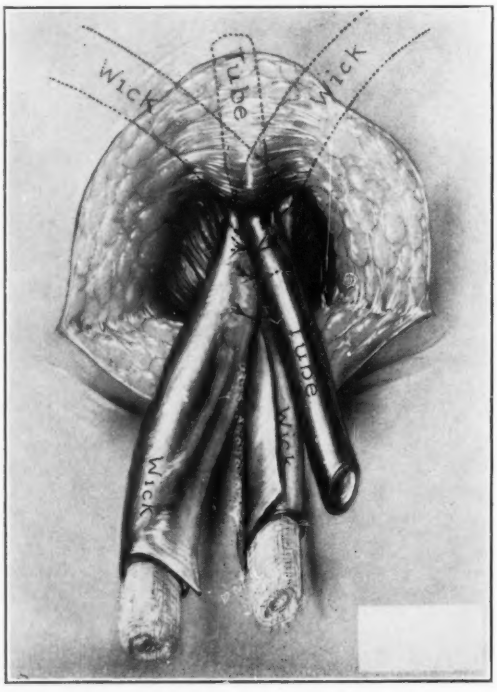

Fig. 6.—Method of prostatoseminal-vesiculocystectomy. Perineal drains are placed.

teriorly and the pubovesical ligaments are divided, but the pubic area is not disturbed because bleeding from the plexus of Santorini is sometimes very difficult to control. By blunt dissection carried laterally, first one and then the other vesical pedicle is isolated, doubly clamped, and tied. This controls the major blood supply. If possible, the seminal vesicles and part of the prostate are then freed from the rectum and extraperitoneal tissues (Fig. 2). The farther down the dissection is carried

TABLE IX

URETEROINTESTINAL TRANSPLANTATIONS FOR CANCER—WITH CYSTECTOMY

REFERENCE IF PREVIOUSLY REPORTED	SEX AGE	INDICATION, OPERABILITY	PREVIOUS TREATMENT IF ANY	OPERATION	NO. OF STAGES	RESULTS, AUTOPSY FINDINGS
C. T.*	M 61	Carcinoma of bladder Operable	Fulgurations	9/13/33: Left ureterointestinal transplant (Higgins' method); abdominal prostatoseminal-vesiculocystectomy; right ureter ligated	1	Died 5 days after operation of generalized peritonitis (left ureter separated from the bowel)
F. B.[60]	M 49	Carcinoma of bladder, invading prostate Inoperable	Left nephrolithotomy 1918, 1933; many fulgurations	9/12/33: Bilateral ureterointestinal transplant (Coffey No. 2 technique); bladder freed down to neck 10/21/33: Perineal prostatoseminal-vesiculocystectomy	2	Died 4 mo. after operation Autopsy: local recurrence
F. H.[61] (Case 5)	M 65	Carcinoma of bladder, invading prostate; bilateral hydronephrosis Inoperable	None	(Patient in uremia) 9/22/33: Bilateral nephrostomy 12/15/33: Simple seven-suture transplant with probe and cautery, left ureter; right ureter tied off; prostatoseminal-vesiculocystectomy	1	Died 2 yr. and 8 mo. after operation, ? cause; 1 yr. after operation had right nephrectomy
M. E.[61] (Case 1)	F 60	Squamous cancer of bladder and urethra Operable	None	12/6/33: Bilateral ureterointestinal transplant (seven-suture method) 1/18/34: Vaginal urethrocystectomy	2	Died 10/18/38, 4 yr. after operation; recurrence vaginally, bilateral pyonephrosis with secondary atrophy of left kidney; after operation had x-ray therapy for recurrence
J. Y.[59] (Case 3)	F 68	Carcinoma of bladder, recurrent Operable	Fulguration	1/6/34: Right ureterointestinal transplant, Coffey No. 3 technique, with extraperitoneal catheter; left ureter to skin; suprapubic cystectomy	1	Died thirteenth day after operation Autopsy: Abscess in vesical space, acute left hydronephrosis with infection; right hydronephrosis; right ureter below the catheter undergoing necrosis

*Previously unreported.

TABLE IX—CONT'D

REFERENCE IF PREVIOUSLY REPORTED	SEX AGE	INDICATION, OPERABILITY	PREVIOUS TREATMENT IF ANY	OPERATION	NO. OF STAGES	RESULTS, AUTOPSY FINDINGS
W. C.[61] (Case 11)	M 72	Carcinoma of bladder, invading prostate Inoperable; pelvis frozen	None	4/26/34: Bilateral ureterointestinal transplant (simple seven-suture method); prostatoseminal-vesiculocystectomy; carcinoma found in perivesical region, not all removed	1	Died eleventh day after operation Autopsy: Acute pyelonephritis, local peritonitis, bronchopneumonia
J. V.[61] (Case 7)	M 63	Squamous carcinoma of bladder, invading prostate Inoperable	Fulguration	6/11/34: Right ureterointestinal transplant (simple seven-suture method); left ureter ligated 8/1/34: Abdominal prostatoseminal-vesiculocystectomy	2	Died fifteenth day after operation from obstruction of left ureter, abscess of left kidney, pelvic abscess
H. S.[61] (Case 10)	M 63	Carcinoma of bladder, recurrent Operable	Many fulgurations	8/27/34: Bilateral ureterointestinal transplant (simple seven-suture with probe and cautery) 10/24/34: Abdominal retroprostatoseminal-vesiculocystectomy	2	Died twenty-fourth day after operation; developed septicemia from pelvic abscess Autopsy: Stenosis, ureteral orifices; *B. coli* septicemia; pelvic abscess
J. M.*	M 69	Carcinoma of prostate Inoperable	None	9/11/34: Bilateral ureterointestinal transplant (seven-suture method with probe and cautery) 10/24/34: Cystectomy only; prostate "frozen" in pelvis	2	Died eleventh day after operation of bronchopneumonia, pelvic abscess; local metastases present; transplant in good condition (autopsy)
L. P. B.[63]	M 54	Carcinoma of bladder Operable	Many fulgurations	10/31/35: Left ureterointestinal transplant (simple seven-suture method) 12/10/35: Right ureterointestinal transplant (simple seven-suture method) and abdominal prostatoseminal-vesiculocystectomy	2	Alive and well 3 yr. after operation; postoperative pyelonephritis

F. E.*	M 66	Carcinoma of bladder Operable	Fulguration	3/22/36: Left ureterointestinal implant (simple seven-suture method with carrier); right ureter placed in intestinal bed 5/6/36: Right ureterointestinal transplant completed; abdominal prostatoseminal-vesiculocystectomy	2	Died fifth day after operation Autopsy: Leak at site of anastomosis on right, pyelonephritis, peritonitis, pneumonia
E. B. H.*	M 58	Carcinoma of prostate, invading bladder Inoperable	None	5/21/36: Left ureterointestinal transplant (simple seven-suture method with carrier) 6/17/36: Right ureterointestinal transplant, same technique, and abdominal prostatoseminal-vesiculocystectomy	2	Died one and one-half yr. after operation, carcinomatosis
A. H.*	F 64	Carcinoma of bladder Operable	Many fulgurations	10/15/36: Left ureterointestinal transplant (simple seven-suture method with probe and cautery) 11/30/36: Right ureterointestinal transplant, same technique; abdominal extraperitoneal cystectomy	2	Died 4/12/39, 2 yr. and 4 mo. after operation of renal insufficiency Autopsy: Bilateral atrophic pyelonephritis, no hydronephrosis or cancer
K. K.*	F 38	Carcinoma of bladder Inoperable	Suprapubic cystotomy one month earlier	11/5/36: Left ureterointestinal transplant (simple seven-suture method with carrier) 12/3/36: Right ureterointestinal transplant, same technique; suprapubic cystectomy	2	Died 4½ mo. after operation, carcinoma in wound
L. A.*	M 54	Squamous carcinoma of bladder Operable	Suprapubic cystotomy 7 mo. earlier, extensive fulguration	12/21/36: Left ureterointestinal transplant (simple seven-suture method with probe and cautery) 2/2/37: Right ureterointestinal transplant (simple seven-suture method with carrier); prostatoseminal-vesiculocystectomy, extraperitoneal	2	Died thirty-seventh day after operation Autopsy: Pelvic abscess, left ureter had pulled away, right ureter normal, pelvic abscess

TABLE IX—CONT'D

REFERENCE IF PREVIOUSLY REPORTED	SEX AGE	INDICATION, OPERABILITY	PREVIOUS TREATMENT IF ANY	OPERATION	NO. OF STAGES	RESULTS, AUTOPSY FINDINGS
F. C.*	F 44	Carcinoma of bladder Operable	Fulguration	12/7/36: Left nephrostomy 1/6/37: Right ureterointestinal transplant (simple seven-suture method with carrier) 2/3/37: Left ureterointestinal transplant, same technique; abdominal extraperitoneal cystectomy	2	Alive 2 yr. after operation; has incisional hernia and suprapubic fistula
N. V.*	M 55	Carcinoma of bladder, invading prostate Operable	Fulguration	1/8/37: Left nephrostomy 2/5/37: Bilateral ureterointestinal transplant (simple seven-suture method with carrier) 3/10/37: Suprapubic prostatoseminal-vesiculocystectomy	2	Died just after operation Autopsy: Massive collapse of lung
A. I.*	M 52	Carcinoma of bladder Operable	Fulguration, x-ray therapy, radium	2/17/37: Left ureterointestinal transplant (seven-suture method with carrier) 3/15/37: Right ureterointestinal transplant, same technique; abdominal cystectomy	2	Alive and well 2 yr. after operation, working hard
L. M.*	F 65	Squamous carcinoma of bladder Operable	Fulguration	4/29/37: Left ureterointestinal transplant (simple seven-suture method with carrier) 5/13/37: Right ureter ligated, vaginoabdominal urethrocystectomy	2	Died 1 yr. and 8 mo. after operation (12/27/38) Autopsy: Recurrence of tumor in pelvis and peritonitis; left kidney in good condition
R. J.*	M 54	Carcinoma of bladder Inoperable	Fulguration	7/14/37: Left ureterointestinal transplant (simple seven-suture method with carrier) 8/20/37: Right ureter ligated, abdominoperineal prostatoseminal-vesiculocystectomy	2	Died 9/16/38, 1 yr. after operation Autopsy: Carcinomatosis; both kidneys free from infection

R. R.*	M 58	Carcinoma of bladder Operable	None	11/26/37: Right ureterointestinal transplant (simple seven-suture method with carrier) 12/23/37: Left ureterointestinal transplant, same technique, abdominoperineal prostatoseminal-vesiculocystectomy	2	Died 10 days after operation Autopsy: Bronchopneumonia; transplant normal; peritoneum clean; wound clean
H. F.*	M 69	Carcinoma of prostate Operable	Suprapubic cystostomy 2 wk. earlier	11/30/37: Right ureterointestinal transplant (simple seven-suture method with carrier) 1/11/38: Left ureterointestinal transplant, same technique, abdominoperineal prostatoseminal-vesiculocystectomy	2	Died 8/16/38, 7 mo. after operation, carcinomatosis
L. S.*	F 55	Carcinoma of bladder Operable	Fulgurations	6/4/38: Left ureterointestinal transplant (simple seven-suture method with carrier) 6/21/38: Right nephrostomy 7/12/38: Total cystectomy (abdominal) 8/8/38: Right nephrectomy.	2	Alive and well 6 mo. after operation
G. C. P.*	M 48	Carcinoma of bladder Operable	Fulguration	11/18/38: Right ureterointestinal transplant (simple seven-suture method with carrier) 12/7/38: Left ureterointestinal transplant, same technique, with abdominoperineal prostatoseminal-vesiculocystectomy	2	Alive and well 1½ mo. after operation
E. G.*	F 47	Epidermoid carcinoma of vagina Operable	Fulguration, x-ray	3/9/39: Left ureterointestinal transplant (seven-suture method with carrier) 3/28/39: Right ureterointestinal transplant with abdominovaginal cystourethrectomy	2	Course after operation uneventful

dorsally, the easier becomes the perineal portion of the procedure. The abdominal wound is closed in layers without drainage (Fig. 3).

The patient is then placed in the usual position utilized in perineal prostatectomy. The prostate is exposed and an extracapsular dissection of the prostate is carried out (Fig. 4). The prostatic urethra is divided, the fascia overlying the seminal vesicles is opened, and this plane of cleavage meets the one developed from above. The prostate, seminal vesicles, and bladder are thereby removed perineally *en masse* (Fig. 5). Two wick drains and a tube drain are placed well up into the extraperitoneal space and the wound is closed (Fig. 6). This procedure ensures excellent dependent drainage of the extraperitoneal dead space.

SUMMARY

1. Total extirpation of the bladder is a feasible operation, particularly in the treatment of vesical cancer.

2. The history of the evolution of this operation is reviewed.

3. The indications for cystectomy are noted.

4. The various methods of diversion of the urinary stream are presented.

5. The technique of total removal of the bladder alone or with adjacent organs is described.

6. The results of total cystectomy are summarized.

7. Twenty-five personal cases are tabulated and analyzed.

8. The technique of abdominoperineal total prostatoseminal-vesiculocystectomy is presented.

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CHOLEDOCHOJEJUNOSTOMY

An Experimental Study After Resection of the Duodenum and Seven-eighths of the Stomach

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THIS report describes the findings in an experimental preparation following resection of the entire duodenum and seven-eighths of the stomach. This was done in order to determine whether such surgical intervention would produce pernicious anemia in dogs. The failure to alter the blood pictures has been reported.[3]

Resection of seven-eighths of the stomach and the entire duodenum necessitates transplantation of the biliary and pancreatic ducts. The pancreatic ducts may be dismissed as relatively unimportant, because in the dog it is almost impossible to keep these ducts from finding their way back into the bowel. The restoration of biliary drainage into the gastrointestinal tract without complicating liver infections seemed, after review of the literature, to be a surgical problem which might prevent a protracted investigation. Experimental reports were mostly discouraging, but Mann and Kawamura[41] found that, after complete duodenectomy and choledochoenterostomy, ten dogs had a survival time of 325 to 555 days. These investigators were interested primarily in whether or not the duodenum was an organ essential for life and, therefore, failed to describe the appearance of their choledochojejunal anastomosis; they made little mention of the presence or absence of liver infection or damage, noting, however, that in some of their animals the common duct was not only dilated, but also markedly infected.

Gage,[17] studying changes in the liver following cholecystogastrostomy and cholecystduodenostomy, first studied forty normal dogs and found histopathologic liver changes in every case. These changes were small areas of necrosis of liver cells with round cell infiltration. These areas at times were rather extensive and in some instances simulated abscess formation. Perivascular infiltration was a common occurrence, but there were no changes in the walls or epithelial lining of the biliary ducts. Fifteen animals were studied fifteen days postoperatively and 53.3 per cent showed no increase in the inflammatory process as compared to normal livers. Seven, or 46.6 per cent, showed slight to moderate increase in pathologic findings when compared to normal livers.

The clinical reports were, however, a basis for assuming that the animal preparations contemplated by us should survive long enough to afford the data desired. Thus, Finsterer,[14] reporting on the permanent

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result after choledochoduodenostomy in forty-two patients, found that 88 per cent were made well from three to twelve years. Bernhard did fifty-eight choledochoduodenostomies with a survival time in two-thirds of the patients of more than fifteen years. DuBose[12] reported excellent clinical results in twenty-one cases. Wangensteen's[66] important contribution in 1928 presented the concept: "Probably only in the event of obstruction to the flow of bile through the new stoma does the potential infection in the bowel become a menace." Oettle[47] has a similar opinion and believes the anastomosis of the biliary tree to the gastrointestinal tract must be wide; that, in a wide anastomosis affording easy filling and emptying of the biliary passages, an infection never follows; and that the unhindered biliary drainage guarantees prompt elimination of invading bacteria as was shown experimentally by Radsiewsky in 1902. Eliason[13] in 1936 concluded that ascending infection was a complication whose frequency was not great enough to discard the operation for "biliary intestinal anastomosis."

These clinical reports convinced us that we should be able to approximate them experimentally with adequate postoperative survival time to answer our experimental problem.

SURGICAL TECHNIQUE

A total of thirty-three dogs was used. The first procedures consisted of either cholecystojejunostomy or choledochojejunostomy after resection of the duodenum and seven-eighths of the stomach. Gastrointestinal continuity was restored by a Polya anastomosis, the gall bladder or the common duct being sutured to the jejunum. The gall bladder was abandoned early in this study, and in the first twenty animals the common duct was fastened by several fine sutures to the jejunum and then a purse-string suture was used to invaginate the bowel around the common duct. Despite satisfactory appearance when the operation was finished, none of these preparations survived long enough for a study of pernicious anemia. Most of the animals died immediately postoperatively from biliary leakage and peritonitis, or two to four months later from obstruction of the common duct.

Beginning with the twenty-first animal, a simpler technique was adopted. All suture material at the site where the common duct traversed the jejunum was eliminated. A small stab wound, about the size of the duct, was made in the jejunum. A single silk suture was inserted into the common duct about 0.5 cm. from the distal end which had been cut from the duodenum. Straight needles were threaded to each end of this suture. They were then inserted through the stab wound into the lumen of the jejunum and finally pushed through the bowel wall about 0.5 cm. apart, approximately 1.5 cm. from the stab wound. When these sutures were tied, the common duct was pulled about 1.5 cm. into the bowel lumen and the jejunum went up and around the duct (Figs.

1 and 2). Immediately after Sept. 20, 1935, when this technique was adopted, the results improved. Seven of the next thirteen animals operated upon by this technique survived for from two to two and three-fourths years. The six deaths were secondary to the following: one animal eviscerated; one common duct pulled out and the animal died from biliary peritonitis; two animals died from peritonitis with the common duct appearing in satisfactory position; and one animal died from an unknown cause, the site of duct transplant appearing satisfactory.

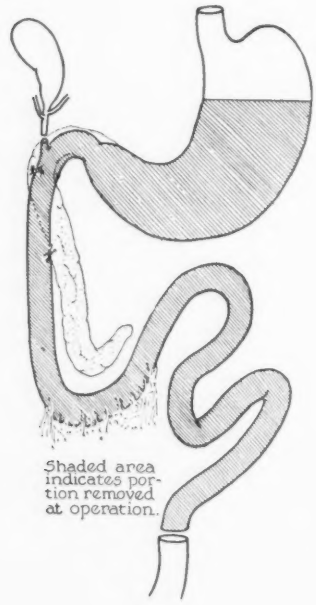

Fig. 1.

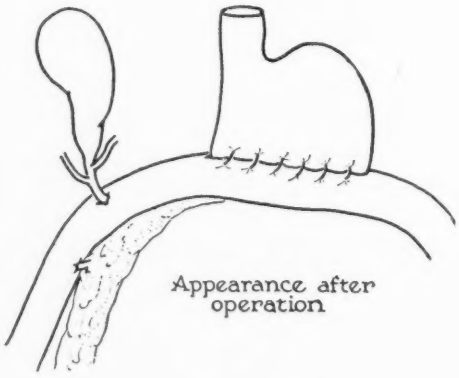

Fig. 2.

As previously reported, none of the animals developed pernicious anemia. They remained in excellent condition for two or more years, after which time three of the dogs died from an infection of the central nervous system which invaded our kennels, occurring not only in this series of animals but also in others not operated upon. The others were sacrificed.

RESULTS

Following modification of surgical technique, the operative mortality immediately decreased and seven out of thirteen animals survived for from two years to two years and nine months. The gall bladder and the biliary tree of one of the animals was visualized from barium given by mouth (Fig. 16).

The essential findings in this study, as shown in Table I, were: (1) the excellent condition of the animals throughout the study; (2) the persistence of a normal blood picture; (3) moderate dilatation of the biliary tree with no obstruction to the distal end of the common duct,

TABLE I

RESULTS

DOG NO.	POST-MORTEM TIME AFTER OPERATION	CAUSE OF DEATH	GASTRIC SECRETION		RED BLOOD CELL CHANGES	SIZE OF BILIARY TREE	GALL-BLADDER* HISTOLOGY	LIVER* HISTOLOGY
			ALCOHOL	HISTAMINE				
1	2¾ yr.	Sacrificed	0	+	None	2.3 times normal	Minor	Minor
2	2¾ yr.	Sacrificed	0	Very little	None	2.4 times normal	Minor	Minor
3	2 yr.	Central nervous infection	0	0	None	2.5 times normal	Negative	Negative
4	2 yr.	Central nervous infection	0	0	None	2.3 times normal	Negative	Negative
5	2 yr.	Sacrificed	0	0	None	1.4 times normal	Minor	Minor
6	2 yr.	Sacrificed	0	0	None	1.6 times normal	Minor	Negative
7	1¾ yr.	Sacrificed	0	0	None	1.4 times normal	Minor	Negative

*For practical purposes the livers were normal histologically and the gall bladder showed no evidence of infection, there being no leucocytic or lymphocytic infiltration; some perilobular fibrosis was present.

which was patent and wide open in all cases; (4) no significant microscopic evidence of active gall-bladder infection (Figs. 9 and 10); (5) even though barium given by mouth to one animal visualized the gall bladder and biliary tree, there was no histologic evidence for liver in-

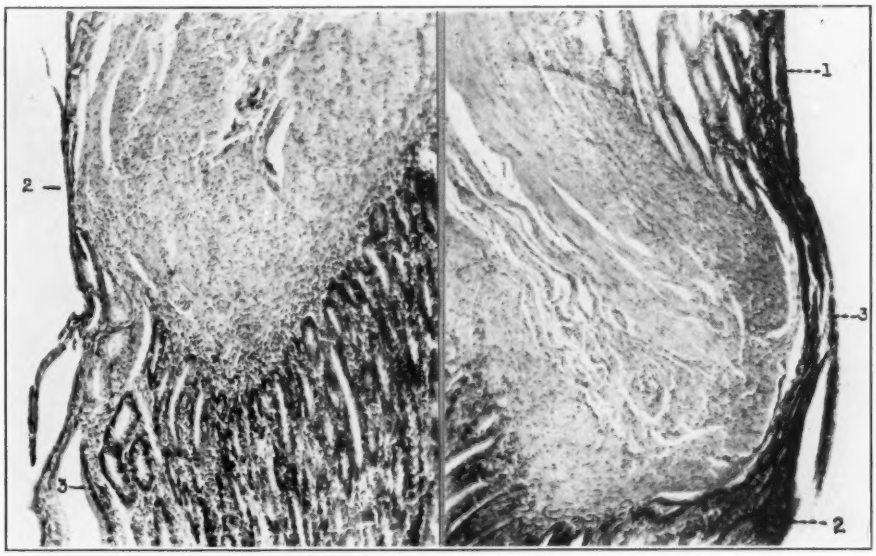

Fig. 3.

Fig. 4.

Fig. 3.—Control Dog ×120. Exact site of juncture between duodenum and common duct. *1*, Duodenum; *2*, common duct; *3*, anastomotic juncture.

Fig. 4.—Dog 2 ×120. Exact site of juncture between jejunum and common duct demonstrating mucosal healing. *1*, Common duct; *2*, jejunum; *3*, anastomotic juncture.

fection or liver damage more than is normal for adult dogs (Figs. 11-14, 16); (6) serial section taken every 10 μ through the site where the common duct entered the jejunum showed mucosal healing, the

Fig. 5. Fig. 6.

Fig. 5.—Dog 6 ×5. Showing common duct transecting jejunum and wide open orifice. Moderate dilatation of the common duct. *1*, Jejunum; *2*, common duct; *3*, anastomotic juncture.

Fig. 6.—Dog 2 ×4. Showing common duct transecting jejunum and wide open orifice. Considerable dilatation of the common duct. *1*, Jejunum; *2*, common duct; *3*, anastomotic juncture.

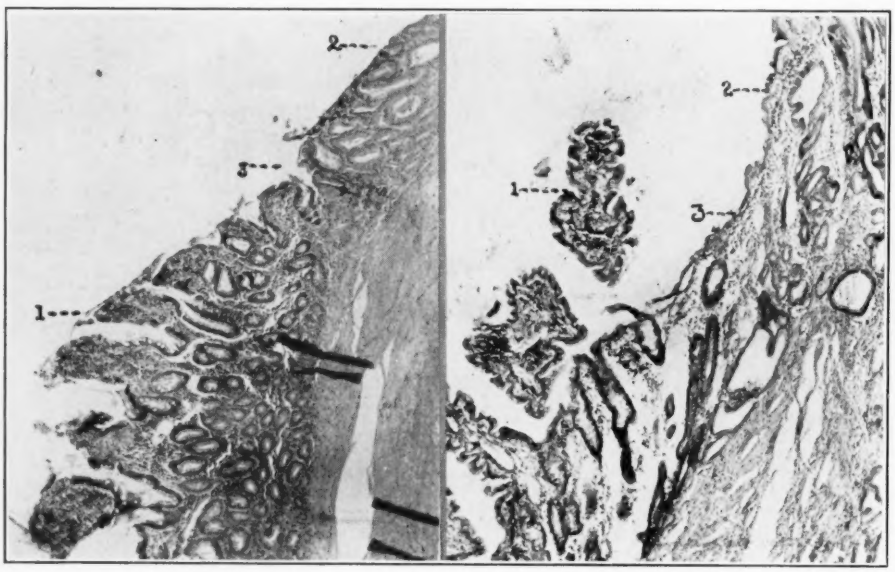

Fig. 7. Fig. 8.

Fig. 7.—Dog 1 ×65. Showing mucosal union between jejunum and common duct. Least satisfactory of all animals. *1*, Jejunum; *2*, common duct; *3*, anastomotic juncture.

Fig. 8.—Dog 3 ×60. Showing mucosal union between jejunum and common duct. *1*, Jejunum; *2*, common duct; *3*, anastomotic juncture.

mucosa of the jejunum healing to the mucosa of the common duct without interposition of connective tissue (Figs. 4-6, and especially Figs. 7 and 8).

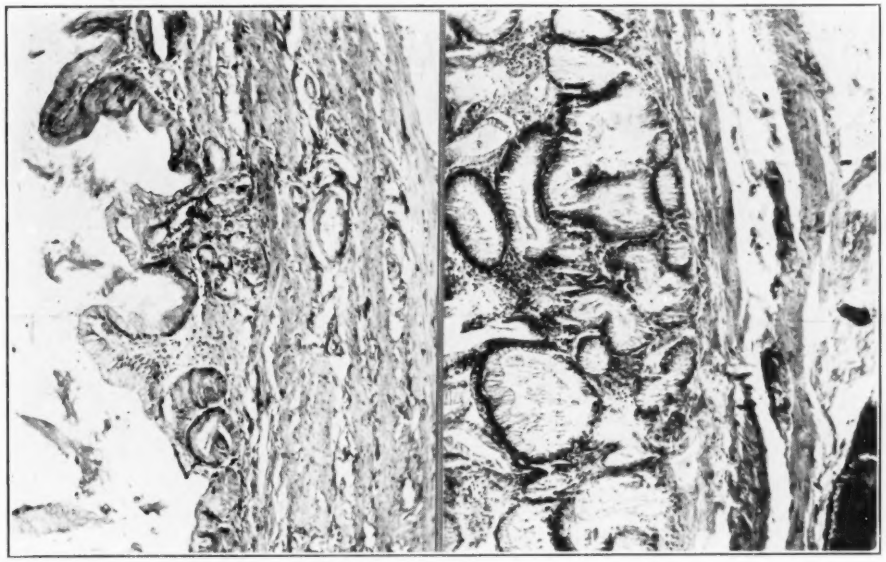

Fig. 9. Fig. 10.

Fig. 9.—Dog 7 ×130. Gall bladder. Practically no dilatation of biliary tree. No histologic evidence of infection.

Fig. 10.—Dog 2 ×130. Gall bladder. Showing little or no inflammatory changes despite marked dilatation of the common duct.

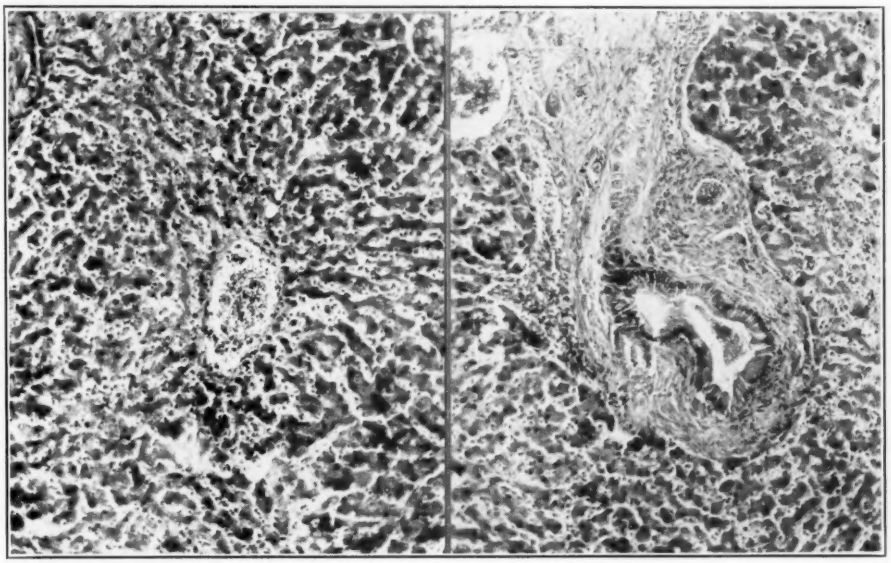

Fig. 11. Fig. 12.

Fig. 11.—Dog 3 ×130. Liver. Liver lobule with central vein. Essentially normal despite finding of barium in the gall bladder.

Fig. 12.—Dog 3 ×130. Liver. Periportal area showing little or no inflammatory changes despite finding of barium in gall bladder.

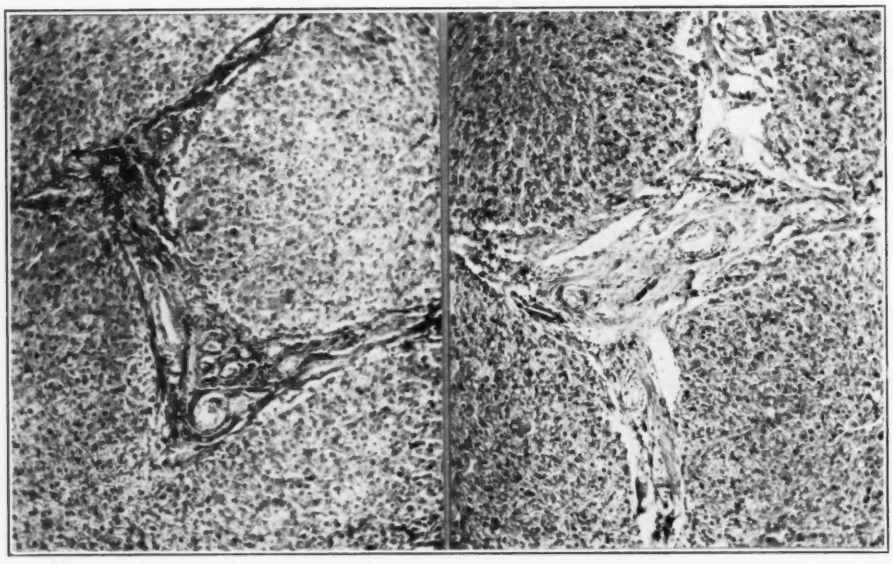

Fig. 13. Fig. 14.

Fig. 13.—Dog 2 ×130. Liver. Animal had marked dilatation of common duct but liver and periportal areas are essentially normal for dog of this age.

Fig. 14.—Dog 7 ×130. Liver. Periportal area which is essentially normal. No evidence of inflammation.

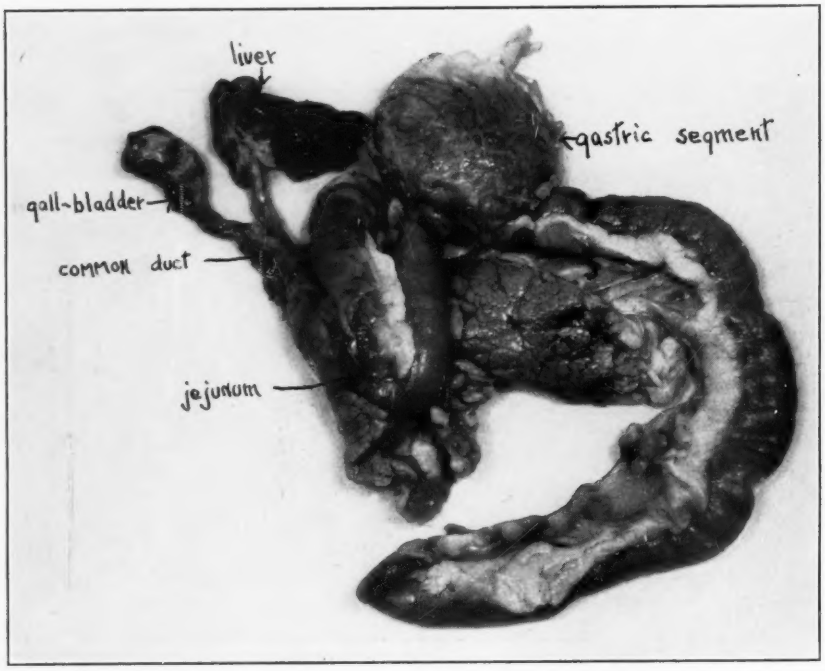

Fig. 15.—Dog 5. Autopsy specimen. Showing little or no dilatation of the biliary tree and very small residual gastric segment.

DISCUSSION

The surgical concept that liver infection is reduced by transplanting biliary ducts to the stomach so that the anastomotic site will be exposed to invasion by acid media believed less septic is not substantiated by these results. The site of anastomosis apparently does not affect the problem of ascending liver infection. This was shown by Sandblom, Bergh, and Ivy[56] and Gentile[20] in that both groups of investigators consistently found infected livers despite the fact that Ivy and associates anastomosed the gall bladder to the duodenum after pyloric exclusion and Gentile used the stomach after gastric resection and Polya-Balfour anastomosis.

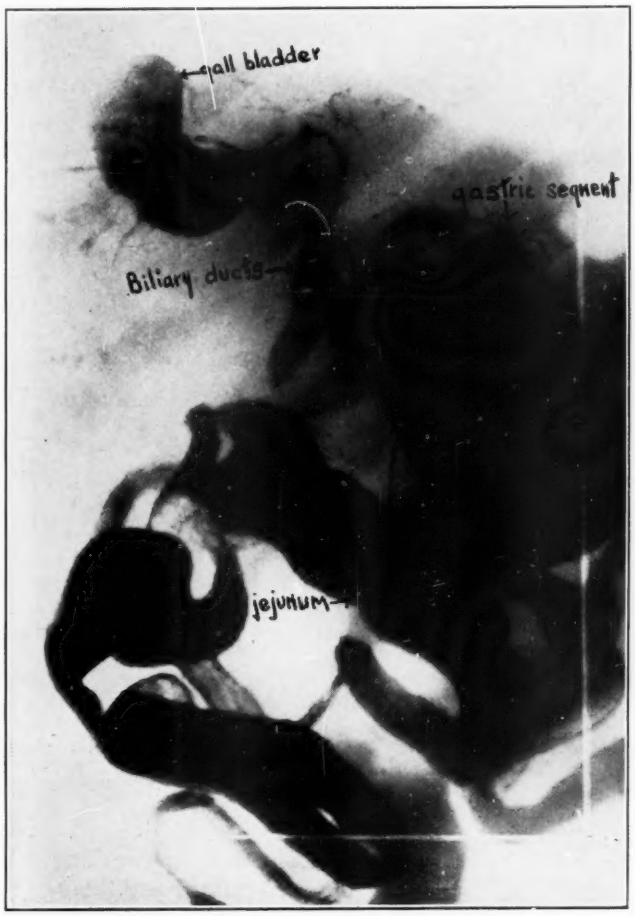

Fig. 16.—Roentgenogram of Dog 3. Showing barium in gall bladder and biliary ducts.

Inasmuch as both hypoacidity and common duct infection were present in our animals, another explanation must be presented for the negligible liver pathology. Our results in confirmation of Wangensteen[66] suggest that the site of anastomosis is not of major importance, but that

its size is. If the anastomotic opening of the biliary tree to the gastrointestinal tract remains persistently patent with a sufficiently large orifice giving adequate drainage, there follows a minimum of stasis with limited ascending infection and little or no liver pathology.

The dilatation of the biliary tree in this series of animals despite patent anastomotic openings would be disturbing but for Branch, Orville and Zollinger,[6] who showed that instrumental dilatation of the papilla of Vater will lead to temporary obstruction and dilatation of the biliary tree. May we not assume that the postoperative inflammatory reaction around the common ducts in our animals led to a temporary biliary obstruction causing dilatation much the same as the instrumental dilatation described by Branch and co-workers?

CONCLUSIONS

1. In the dog, choledochojejunostomy is feasible after gastric and duodenal resection.

2. Such animals remain in excellent condition for as long as two and three-fourths years.

3. Pernicious anemia does not develop.

4. Modification of the technique used for choledochojejunal anastomosis in our animals was followed by: (a) reduction in postoperative mortality; (b) decreased incidence of common duct obstruction; (c) choledochojejunal anastomosis with mucosal healing, having practically no scar or stenosis from connective tissue formation; and (d) histologic evidence of minimum gall-bladder and liver infection with histologic changes not exceeding normal for adult dogs.

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THE EFFECT OF GALL-BLADDER DISEASE ON THE ELECTROCARDIOGRAM

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ATTEMPTS to answer the question—Can a diseased gall bladder cause myocardial degeneration?—have been productive of diverse opinions in the literature.* These multiple reports and statements on the relation of gall-bladder disease to heart disease are extremely difficult to evaluate clinically. In those reports, for instance, of the presumed cure of angina pectoris following the removal of a diseased gall bladder, it is impossible to exclude the probability that the disappearance of the attacks was merely the result of a spontaneous remission in the course of the angina pectoris. Possibly the pain was not cardiac in origin but was referred from the diseased gall bladder to the heart.

Recently, Fitz-Hugh, Jr., and Wolferth[1] reported reversible electrocardiographic changes in six patients with gallstones who were surgically treated. These patients all exhibited cardiac symptoms, chiefly anginoid effort types, and all were found to have abnormal electrocardiographic tracings, chiefly flat or inverted T-waves in the first two leads. In all the cases clinical symptoms were benefited and there was a return toward normal of the electrocardiographic deviations within a few weeks to a few months after the surgical removal of the gallstones.

The significance of their electrocardiographic observations is evident. If a diseased gall bladder can produce an abnormal tracing, a more critical clinical application of electrocardiographic deviations must be applied. We determined to repeat their studies. Accordingly electrocardiograms were made before and after surgery on patients with gall-bladder disease. Twenty-one patients underwent surgery. All had chronic cholecystitis with cholelithiasis. There was only one male patient. The ages ranged from 21 to 68 years. Six patients had essential hypertension as evidenced by a persistent elevation of the blood pressure. Only two of this group had roentgenologic evidence of cardiac enlargement. One patient with cardiac enlargement but a blood pressure of 148/84 was likewise considered to have hypertensive heart disease. None of the other etiologic types of heart disease was encountered. Chest pain at all significant of angina pectoris was not complained of by any of the patients. Only one patient gave a history of severe dyspnea on mild exertion and she had hypertensive heart disease.

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*No attempt is made to list all the literature. This has been done by numerous authors. See Laird, S. M.: Brit. M. J. 1: 884, 1938.

Significant abnormal preoperative electrocardiograms were found in four cases. Three had electrocardiograms which became normal from six to eight days after operation. One of these three patients had hypertensive heart disease so that her preoperative records may have been transiently abnormal as occasionally occurs in hypertensive individuals. The one case that showed no change after operation had hypertensive heart disease. There remain two patients whose only evidence of heart

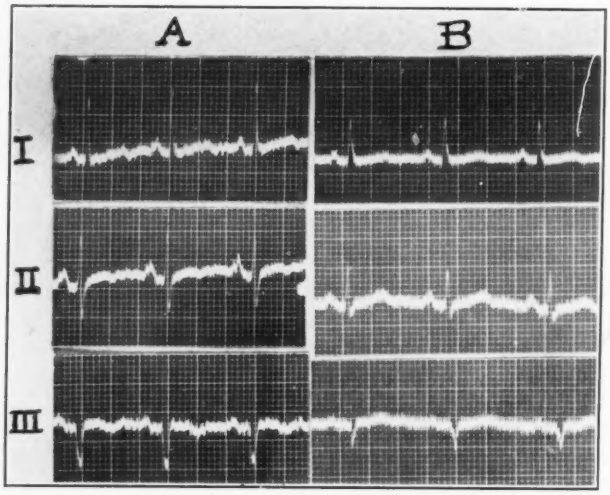

Fig. 1.—Electrocardiograms of Mrs. C., white, aged 40 years. *A*, made five days before cholecystectomy, show T-wave inverted in Lead II. *B*, made eight days postoperatively, show no abnormalities.

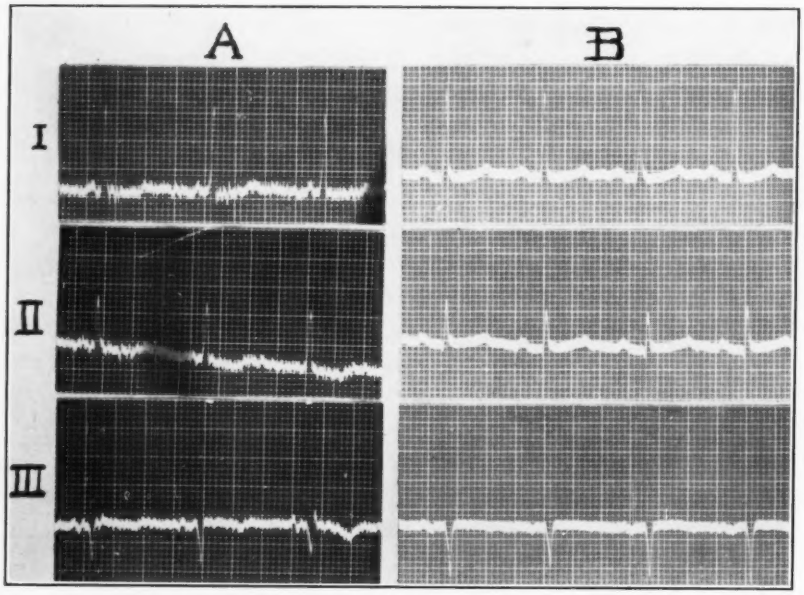

Fig. 2.—Electrocardiograms of Mrs. R., white, aged 53 years. *A*, made eight days before cholecystectomy, shows depression of S-T interval and T-wave inversion in Lead II. *B*, made six days postoperatively, are normal.

disease was an abnormal electrocardiogram, which tracing became normal after the removal of gallstones and a diseased gall bladder.

The electrocardiograms of one of these patients which were made five days before a cholecystectomy was performed are shown in Fig. 1*A*. The T-wave in Lead II is inverted. The tracings shown in Fig. 1*B* were made eight days after the operation. The T-wave in Lead II is now upright and has remained normal in repeated examinations. This patient was a white woman, 40 years of age.

The electrocardiograms of the other case, a 53-year-old white woman, made eight days preoperatively show (Fig. 2*A*) in Lead II depression of the S-T interval with the T-wave inverted and of low voltage. The tracings made six days postoperatively (Fig. 2*B*) are normal.

COMMENT

As noted by Fitz-Hugh and Wolferth, the electrocardiographic deviations consisted for the most part of T-wave changes in significant leads. Slurring and notching of the QRS complexes, marked elevation or depression of the S-T interval or coronary occlusion types of T-waves were not noted.

The answer to the question—Can a diseased gall bladder cause myocardial degeneration?—must await pathologic study. The present report merely confirms that of Fitz-Hugh and Wolferth that gall-bladder disease per se can produce abnormal electrocardiograms which revert to normal after surgical amelioration of the gall-bladder disease. The practical application of these observations in regard to the interpretation of T-wave changes in electrocardiograms is evident.

The rapid reversal to normal in the cases cited above would indicate a toxic effect on the myocardium of the diseased gall bladder.

There was no correlation between the clinical and pathologic severity of the gall-bladder disease and the T-wave changes. Normal records were obtained in patients with the worst pathologic gall bladders.

SUMMARY

1. Gall-bladder disease may produce abnormal T-wave changes in the electrocardiogram.

2. After removal of the diseased gall bladder, the electrocardiogram can become normal.

3. There is no correlation between the clinical and pathologic severity of the gall-bladder disease and the electrocardiographic abnormalities.

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THE EFFECT OF TRAUMA TO THE LIVER ON THE PLASMA PROTHROMBIN*

An Experimental Study

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THAT an operative procedure is a significant factor in the production of the hemorrhagic tendency associated with jaundice is indicated by the fact that the overwhelming majority of patients who bleed do so after operation. The underlying defect which is responsible for the hemorrhage in jaundice is a deficiency of plasma prothrombin, and a number of observations indicate that the liver is intimately concerned in the maintenance of the normal level of this substance. Smith, Warner, and Brinkhous[1] have shown that a precipitous fall in plasma prothrombin occurred in hepatic damage incident to chloroform anesthesia in dogs, and Warner,[2] after removing approximately 65 per cent of the liver of the rat, demonstrated a fall in plasma prothrombin which returned to normal in about two and one-half weeks. Andrus, Lord, and Moore[3] have shown that total removal of the liver in dogs was followed by a fall in plasma prothrombin to less than 20 per cent of normal within ten hours.

Since, during most operative procedures on the biliary tract, mechanical trauma to the liver is unavoidable, it has seemed important to study experimentally the level of plasma prothrombin following mechanical trauma to the liver.

METHODS

Normal healthy dogs weighing between 25 and 40 pounds were selected for the operative procedures. Intravenous nembutal anesthesia was employed. Plasma prothrombin was determined by the method of Warner, Brinkhous, and Smith.[4] Each animal was anesthetized, and under aseptic precautions the peritoneal cavity was opened through an upper right rectus incision. The various lobes of the liver were then gently massaged between the fingers and thumb for twenty-five minutes. The massage was not sufficiently severe to cause either tearing of the capsule or hemorrhage. The abdomen was then closed in layers. Blood samples for the determination of plasma prothrombin were drawn before the operation, at the cessation of trauma to the liver, two hours postoperatively, and at suitable periods thereafter. The general postoperative behavior of the dogs was in no way different from that of animals subjected to a simple exploratory laparotomy.

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RESULTS

The results of the experiments are shown in Table I. All designations of plasma prothrombin are in percentages of the preoperative value. In all but two experiments there was an immediate fall in plasma prothrombin to an average of 71 per cent of the preoperative level. During the ensuing twenty-four hours, the level remained in the neighborhood of 70 per cent. Subsequently, sometime between the forty-eighth and one hundred and forty-fourth postoperative hour, a return to the preoperative level occurred. Experiments 6 and 7 were performed on the same animal on different occasions. On one of those occasions there was a slight rise in plasma prothrombin after trauma to the liver, while in the other a significant fall occurred, although its appearance was slightly delayed.

Fig. 1 shows in a solid line a composite curve of the seven experiments in which the liver was traumatized. The average fall in plasma prothrombin following trauma to the liver was to 75 per cent of the original level, and six days elapsed before the plasma prothrombin returned to the preoperative level.

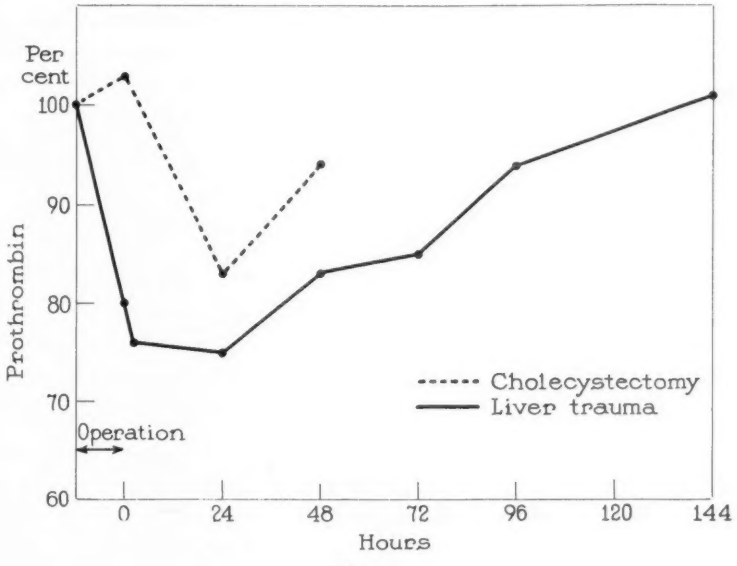

Fig. 1.

The broken line in Fig. 1 shows the average values for the plasma prothrombin of two dogs which were subjected to a cholecystectomy and ligation and division of the common bile duct. Although the liver was handled as little and as gently as possible, a 15 and 20 per cent fall in plasma prothrombin occurred at the end of twenty-four hours, with a partial return to normal in forty-eight hours. Simple laparotomy with the peritoneal cavity exposed for twenty-five minutes resulted in no change in the plasma prothrombin. Likewise, operations of the magni-

tude of a reverse Eck fistula[5] and a gastroenterostomy caused no fall in the plasma prothrombin.

Four dogs subjected to massage of the spleen and kidney have shown an average fall in plasma prothrombin to 75 per cent of the preoperative

TABLE I

PLASMA PROTHROMBIN FOLLOWING TRAUMA TO THE LIVER

EXPERIMENT NO.	DOG NO.	PLASMA PROTHROMBIN								
		PREOPERATIVELY	AT END OF TRAUMA	HOURS POSTOPERATIVELY						
				2	24	48	72	96	120	144
1	454	100%	66%		64%		84%	101%		
2	454	100%	66%	70%	64%	101%				
3	271	100%	80%	61%	75%	90%				100%
4	P-21	100%	73%	76%	80%	78%	85%	91%		106%
5	P-22	100%	72%	70%	67%	73%	69%	97%		
6	P-19	100%	103%	108%		95%				110%
7	P-19	100%	99%	79%	77%	73%	72%		81%	87%
Average		100%	80%	76%	75%	83%	85%	94%		101%

level within twenty-four hours. In contrast to the recovery period of six days after massage of the liver, the plasma prothrombin after massage of the spleen and kidney returned to the preoperative level within forty-eight hours. From these findings it is evident that general tissue injury is followed by a prompt and significant fall in plasma prothrombin and that this fall is not peculiar to liver injury. However, the marked difference in the rate of recovery of the plasma prothrombin is further evidence that the liver is more intimately concerned in the elaboration of plasma prothrombin than is the spleen or kidney.

SUMMARY

The results indicate that trauma to the liver, such as might be necessary in difficult operations on the biliary tract, causes a profound decrease of the plasma prothrombin. Since the majority of patients with jaundice already have a decreased plasma prothrombin, such a postoperative fall may depress the prothrombin to the bleeding level of about 20 per cent. It is probable that these observations offer one explanation of the fact that the hemorrhage in jaundiced patients, when it occurs, does so after an operative procedure.

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THE EFFECT OF HEPATECTOMY ON THE PLASMA PROTHROMBIN AND THE UTILIZATION OF VITAMIN K*

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CERTAIN EXPERIMENTAL work has suggested that the liver may be the essential organ in the maintenance of the level of the plasma prothrombin. Thus, in 1937, Smith, Warner, and Brinkhous[1] showed that hepatic damage caused by prolonged chloroform anesthesia was followed by a significant fall in plasma prothrombin and that about a week elapsed before the normal level was regained. Warner[2] noted a similar decrease of the plasma prothrombin after removal of about 65 per cent of the liver of the rat, with a return to normal in about 2½ weeks. And finally Lord[3] recently has shown that even gentle massage of the liver over a period of twenty-five minutes produces a fall of 25 per cent in the plasma prothrombin level which is recovered from after 6 days.

Extending this work, we have performed complete hepatectomy in six dogs using the techniques of Mann[4] and of Markowitz, Yater, and Burrows.[5] This was followed by a precipitate fall in the plasma prothrombin of 45 per cent within one hour after operation which continued until by eight or ten hours it had reached 20 per cent of normal or lower (Table I). In two animals which survived longer than ten hours the plasma prothrombin fell to 5 per cent of normal. At autopsy, petechiae and ecchymoses were noted as early as 11 hours after hepatectomy.

TABLE I

TYPE OF EXPERIMENT	PLASMA PROTHROMBIN IN PERCENTAGE OF NORMAL AT HOURS INDICATED					
	0	1	5½	7½	9	10
I. Instillation of vitamin K	23*	19	29	37	44	47
II. Hepatectomy	100	57	38	20	17	14
III. Hepatectomy and instillation of vitamin K	100	54	32	22	17	15

*Each figure is the average of two experiments.

Contributing to the abrupt initial fall in the plasma prothrombin may be the factor of trauma to the liver incident to the operation, but the

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greater decrease and its continuance would seem to be directly connected with the absence of this organ.

It is now well recognized that, in the absence of hepatic damage, an animal or patient with a lowered plasma prothrombin responds to the administration of vitamin K and bile salts by a rise in the level of the prothrombin of the plasma within a few hours. Thus, in two of our animals whose plasma prothrombin had become markedly decreased because of long-standing biliary fistulas, the instillation of 10,000 units of vitamin K and 2 gm. of bile salts into the duodenum was followed by a rise of the plasma prothrombin as determined by the method of Warner, Brinkhous, and Smith[6] from the level of 23 per cent to 47 per cent in ten hours.

To study the effect of hepatectomy on the utilization of vitamin K, two animals of this series whose livers were removed received similar instillations of 10,000 units of vitamin K (Klotogen) and 2 gm. of bile salts (bilein) into the duodenum at operation, but this failed to alter the typical curve of decrease in the plasma prothrombin level seen in hepatectomized dogs without such medication.

SUMMARY

The precipitate and continuous fall in the plasma prothrombin which follows hepatectomy is evidence that the liver, under normal conditions, is concerned in the continuous formation of prothrombin. Further evidence that the liver is the organ which, in the presence of vitamin K, forms prothrombin is found in the fact that instillation of a large amount of this vitamin during operation fails to alter the typical curve of decrease in the plasma prothrombin after hepatectomy.

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THE IMPERMEABILITY OF THE VIABLE OBSTRUCTED BOWEL OF DOGS TO *CLOSTRIDIUM BOTULINUM* TOXIN*

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THE absorption of toxic substances from the obstructed bowel is believed, by some investigators, to be the significant factor in producing death in intestinal obstructions. It would appear, however, that absorption of substances to which the normal bowel is permeable, via the mesenteric veins, is retarded in bowel obstruction as shown by Wangensteen and his associates.[3, 5] An increased rate of absorption via the mesenteric lymphatic channels was observed by Sperling and Wangensteen,[6] but this factor did not appear to be reflected in the mortality after division of the mesenteric lymphatic pedicle. Increase of intraluminal pressure which impairs the viability of the bowel, thus leading to greater mural permeability and opening up a new avenue of absorption, the transperitoneal, was believed by Wangensteen[8] to be the important agency in the cause of death in experimental ileal obstructions and in low obstructions in man.

Haerem, Dack, and Dragstedt[1] injected *Clostridium botulinum* toxin into closed intestinal loops of dogs and reported that abnormal absorption could be demonstrated in the blood of such animals, while no evidence of absorption was obtained from the bowel of normal animals. In the light of recent denials of increased absorption in viable intestinal loops in bowel obstructions, the observations of Haerem and her associates have more than ordinary significance. Their experiments suggest that the viable obstructed bowel may be permeable to substances to which the bowel wall is normally impermeable.

The following experiments were undertaken to investigate the extent and manner of absorption of botulinum toxin in experimental intestinal obstructions as reported by Haerem and her associates.

METHOD

The following experiments were devised to establish the permeability of the normal dog bowel wall to *Clostridium botulinum* toxin: (1) the toxicity of dog's blood after intravenous toxin administration; (2) the

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passage of the toxin into the peritoneal fluid after intravenous administration; (3) the passage of toxin into the blood after intraperitoneal administration, and (4) the impermeability of the normal Biebl loop to the toxin.

In all the experiments dogs were used. The presence of botulinum toxin was determined by the presence or absence of intoxication of mice. All injections of blood, peritoneal fluid, Berkefeld-filtered intestinal contents, or toxin into mice were of 0.2 c.c. quantities, measured in a 1 c.c. tuberculin syringe fitted with a 25 gauge needle, injected intraperitoneally into mice weighing 20 to 25 gm., in each instance in duplicate. Before the toxin was used in any experiment, its potency was determined by injection as described, the time being noted when the mice died. In each experiment, the potency test was performed; thus, "mouse potency, 2 hours" would indicate that duplicate mice injected intraperitoneally with 0.2 c.c. toxin died two hours after the injection.

All surgery was performed with surgical asepsis under intravenous pentobarbital sodium anesthesia (35 mg./kg.). Where peritoneal fluid samples were to be obtained, preliminary omentectomy was performed twenty-four hours before to facilitate free drainage of peritoneal fluid through a catheter with multiple perforations sutured to the anterior parietal peritoneum and emerging from the abdomen through a stab wound.

The closed jejunal loops in the dogs were made according to a method modified from Stone, Bernheim, and Whipple,[7] the proximal closed end in every case being about 5 to 10 cm. distal to the ligament of Treitz, and the distal closed end being at a variable distance from it, the exact length being stated in each protocol. All injections into the bowel were made at least twenty-four hours after the particular segment had been brought to the outside of the abdomen and covered with skin, the familiar Biebl loop, which has the appearance of a satchel handle on the abdomen. Each dog equipped with a peritoneal catheter was dressed in a canvas vest through which the forelegs were placed and which was laced along the animal's back.

EXPERIMENT 1.—Mouse potency, 2 hours. A small dog was given 20 c.c. of toxin by stomach tube with no symptoms of intoxication appearing at any time. The animal's peripheral venous blood aspirated at 3, 7, 10, and 20 hours after intubation was not toxic for mice (No. 1, Table I).

EXPERIMENT 2.—Mouse potency, 1 hour, 45 minutes. The control blood and peritoneal fluid of a previously omentectomized medium sized dog were not toxic for mice. Forty cubic centimeters of the toxin were then administered to the dog by stomach tube, and postinjection samples of blood and peritoneal fluid obtained at intervals of 2, 6, 10, 14, and 24 hours were mouse-tested and were found to be nontoxic (No. 2, Table I).

EXPERIMENT 3.—Mouse potency, 2½ hours. The control blood and peritoneal fluid of a previously omentectomized small dog were not toxic for mice. Twenty cubic centimeters of toxin were then administered by stomach tube and samples

of blood and peritoneal fluid obtained at intervals of 2, 7, 11, and 14 hours after intubation were mouse tested and found to be nontoxic. Twenty-four hours after intubation, the dog was sacrificed by intravenous injection of 5 c.c. ether, and the contents of the stomach, jejunum, and ileum were Berkefeld-filtered and mouse-tested. These mice succumbed in 30 to 36 hours (No. 3, Table I).

TABLE I

TOXICITY FOR MICE OF BLOOD AND PERITONEAL FLUIDS OF DOGS INJECTED WITH *Cl. botulinum* TOXIN BY VARIOUS ROUTES

NO.	ROUTE OF TOXIN ADMINISTRATION	TIME AFTER TOXIN ADMINISTRATION*	TOXICITY OF BLOOD†	TOXICITY OF PERITONEAL FLUID	EFFECT ON DOG
1	Stomach tube	Control	0 0		No effect
		3 hr.	0 0		
		7 hr.	0 0		
		10 hr.	0 0		
		20 hr.	0 0		
2	Stomach tube	Control	0 0	0 0	No effect
		2 hr.	0 0	0 0	
		6 hr.	0 0	0 0	
		10 hr.	0 0	0 0	
		14 hr.	0 0	0 0	
		24 hr.	0 0	0 0	
3	Stomach tube	Control	0 0	0 0	Sacrificed 24 hr. after intubation
		2 hr.	0 0	0 0	
		7 hr.	0 0	0 0	
		11 hr.	0 0	0 0	
		14 hr.	0 0	0 0	
	Post-mortem gastrointestinal contents nonspecific for toxin				
4	Mesenteric vein	Control	0 0		Died 35 hr. after injection
		2 hr.	+ +		
		5 hr.	+ +		
		8 hr.	+ +		
5	Leg vein	Control	0 0		Died 11 to 12 hr. after injection
		1 hr.	+ +		
		3 hr.	+ +		
		7 hr.	+ +		
		10 hr.	+ +		
6	Leg vein	Control	0 0	0 0	Died 18 to 20 hr. after injection
		2 hr.	+ +	? ?	
		6 hr.	+ +	+ +	
		10 hr.	+ +	+ +	
		14 hr.	+ +	+ +	
7	Intraperitoneal	Control	0 0		Died 15 hr. after injection
		4 hr.	+ +		
		8 hr.	+ +		
		11 hr.	+ +		
8	Normal Biebl loop	Control	0 0		No effect
		2 hr.	0 0		
		6 hr.	0 0		
		8 hr.	0 0		
		20 hr.	0 0		
9	Closed jejunal loop (Biebl)	Control	0 0	0 0	Died 16 hr. after injection
		8 hr.	0 0	0 0	
	Intraluminal pressure, 48 cm. water; hemorrhagic necrosis of loop at autopsy				

*Samples aspirated before toxin administration are designated "control."

† +, death of mouse; 0, mouse alive.

The first three control experiments would indicate that dogs are unharmed by botulinum toxin given by mouth, the blood and peritoneal fluid being free of demonstrable toxin. If any toxin was present in the intestinal contents of these animals the following day, it was too small to be detected by the mouse test.

EXPERIMENT 4.—Mouse potency, 3 hours. The control blood of a small dog was nontoxic for mice. A mesenteric vein was then carefully packed off and a No. 25 gauge hypodermic needle inserted into the vessel and directed toward the root of the mesentery. A syringe containing 4 c.c. of toxin was carefully attached and the toxin was slowly injected. There was no contamination of the peritoneum with toxin or mesenteric blood. The abdomen was then closed and samples of peripheral blood obtained at 2, 5 and 8 hours after the mesenteric injection were mouse-tested; these mice died within a few hours of botulinum intoxication. The dog died of intoxication 35 hours after laparotomy (No. 4, Table I).

EXPERIMENT 5.—Mouse potency, 2½ hours. After aspirating a sample of peripheral venous blood as control, 5 c.c. of toxin were injected intravenously into a medium-sized dog and blood samples were aspirated for mouse testing at intervals of 1, 3, 7 and 10 hours after the injection, and found to be lethal. Death of the dog occurred 11 to 12 hours after toxin administration (No. 5, Table I).

Control Experiments 5 and 6 confirm the lethal effect of the toxin on the dog when entrance into the blood stream has been gained.

EXPERIMENT 6.—Mouse potency, 2½ hours. Six cubic centimeters of toxin were injected intravenously into a previously omentectomized medium-sized dog whose blood and peritoneal fluid obtained before injection were negative by mouse test. Samples of blood and peritoneal fluid were mouse tested 2, 6, 10, and 14 hours after administration of the toxin. All the postinjection blood samples contained toxin. The 2-hour peritoneal fluid sample, however, had its lethal effect on the mice delayed for 20 to 30 hours, while the 6, 10, and 14 hour samples killed the mice in 8 to 16 hours. The dog died 18 to 20 hours after toxin administration (No. 6, Table I).

Control Experiment 6 would indicate that the blood and peritoneal fluid are in chemical equilibrium as regards intravenously injected toxin. This is also demonstrated in Experiment 7.

EXPERIMENT 7.—Mouse potency, 2½ hours. The control blood sample from a small dog was innocuous for mice. Twenty cubic centimeters toxin were injected intraperitoneally and blood samples aspirated at 4, 8, and 11 hours after administration of the toxin. These were all mouse-positive. The blood obtained 4 hours after toxin injection killed mice in three hours. Death of the dog occurred in 15 hours (No. 7, Table I).

EXPERIMENT 8.—Mouse potency, 1½ to 2 hours. The control blood from a large dog with a high, unobstructed jejunal Biebl loop was negative by mouse test. Twenty-five cubic centimeters of toxin were injected into the lumen of the loop. Blood samples were aspirated at 2, 6, 8, and 20 hours and found to be nontoxic for mice (No. 8, Table I).

Control Experiment 8 shows that toxin is not demonstrable in dog's blood by the mouse method after injection directly into the jejunum. In

the following experiments the permeability of the closed loop wall to botulinum toxin was studied in relation to the intraluminal pressure which was determined directly by a needle inserted directly into the Biebl loop and measured by the level of a sustained single fluid column.

EXPERIMENT 9.—A 45 cm. closed jejunal loop was constructed in a medium-sized dog. A Biebl loop was made of the midportion of this; omentectomy was performed and peritoneal drainage established. Twenty-four hours later the pressure in the loop was 48 cm. water and samples of blood and peritoneal fluid were mouse-tested as controls and found to be negative. One hundred and five cubic centimeters of fluid intestinal contents were aspirated from and 5 c.c. botulinum toxin (mouse potency, 2 hours) injected into the Biebl loop. Peritoneal fluid and blood obtained from the dog 8 hours later were innocuous for mice. The dog died 40 hours after operation (16 hours after toxin injection). The autopsy showed the obstructed loop to have undergone hemorrhagic necrosis, with areas of gangrene being most prominent along the antimesenteric border (No. 9, Table I).

This experiment shows that botulinum toxin is not absorbed from obstructed loops directly into the general circulation nor into the peritoneum even at 48 cm. water pressure. The rapid rise of pressure in obstructed loops of the upper reaches of the gut, as compared with low intestinal loops, has been explained by Morton and Sullivan[2] as probably being due to increased secretion. Sperling[4] found that in a dog intracolonic pressure of 30 cm. water sustained for 24 hours caused hemorrhagic damage to the wall; at 50 cm. water pressure, perforation occurred. Damage from distention, therefore, is to be reckoned with seriously. In Experiment 9, therefore, the final samples of blood and peritoneal fluid were taken just before seepage, perforation, and peritoneal contamination had occurred.

EXPERIMENT 10.—A large dog was prepared with a Biebl loop of closed jejunum and peritoneal catheter exactly as in Experiment 9. Twenty-four hours later the intrajejunal pressure was 37 cm. water, when blood and peritoneal fluid were found to contain no toxin. The unfiltered loop contents killed mice in 4 to 10 hours. Five cubic centimeters botulinum toxin (mouse potency, 1½ hours) were injected directly into the loop, and samples of blood, peritoneal fluid, and unfiltered loop contents were mouse tested at 2 hours and 11½ hours after toxin administration. No toxin was demonstrated in the 2-hour samples of blood and peritoneal fluid; the unfiltered intestinal contents killed the mice rapidly. The 11½-hour blood, peritoneal fluid, and intestinal contents rapidly killed mice. The dog died 36 hours after operation with hemorrhagic necrosis of the isolated loop (No. 10, Table II).

EXPERIMENT 11.—A small dog was prepared exactly as in Experiments 9 and 10, except that the closed loop was 35 cm. in length and the original contents of the loop were removed by brief perfusion with warm water. Twenty-four hours later the intrajejunal pressure was 39 cm. water, when blood, peritoneal fluid, and Berkefeld-filtered loop contents were negative on mouse test. Three and one-quarter cubic centimeters botulinum toxin (mouse potency 1½ hours) were injected directly into the Biebl loop, and samples of blood, peritoneal fluid, and Berkefeld-

TABLE II

TOXICITY FOR MICE OF BLOOD, PERITONEAL FLUID, AND CLOSED JEJUNAL LOOP CONTENTS OF DOGS INJECTED WITH *Cl. botulinum* TOXIN DIRECTLY INTO THE EXTERIORIZED LOOP

NO.	TIME AFTER TOXIN ADMINISTRATION*	INTRALUMINAL PRESSURE	TOXICITY OF BLOOD	TOXICITY OF PERITONEAL FLUID†	TOXICITY OF LOOP CONTENTS
10	Control	37 cm. H_2O	0 0	0 0	+ +
	2 hr.		0 0	0 0	+ +
	11½ hr.		+ +	+ +	+ +
					(Loop contents injected unfiltered)
11	Control	30 cm. H_2O	0 0	0 0	0 0
	2 hr.	----	0 0	0 +	0 +
	12 hr.	34 cm. H_2O	0 0	+ +	0 0
	25 hr.	34 cm. H_2O	0 0	0 +	0 0
	47 hr.	91 cm. H_2O	0 0	0 +	0 0
					(Loop contents injected after Berkefeld filtration)
12	Control		0 0	0 0	0 0
	2 hr.		0 0	0 0	+ +
	12 hr.		0 0	0 0	0 +
	24 hr.		0 0	0 0	0 0
					(Loop contents injected after Berkefeld filtration)

*Samples aspirated before toxin administration are designated "control."
† +, death of mouse; 0, mouse alive.

filtered intestinal contents were mouse tested at 2, 12, 25, and 47 hours after administration of the toxin (No. 11, Table II). The contents of the isolated loop previously injected with toxin, as well as the dog's blood after toxin injection, were not toxic for mice even 71 hours after obstruction.

Apparently, the toxin is destroyed between 12 and 25 hours after its injection into the loop. Since no toxin could be demonstrated in the blood at any time, although the peritoneal fluid was questionably toxic, the experiment was repeated, culturing the liquids injected into the mice and culturing the heart blood and peritoneal fluid of the mice immediately after death.

EXPERIMENT 12.—A medium-sized dog was prepared with a Biebl loop of closed jejunum, 40 cm. in length, as described in Experiment 9. Twenty-four hours later samples of blood, peritoneal fluid, and Berkefeld-filtered loop contents were cultured in liver peptone and dextrose broth and injected into mice. Twenty cubic centimeters botulinum toxin (mouse potency, 2 to 6 hours) were then injected directly into the Biebl loop, and samples of blood, peritoneal fluid, and filtered loop contents aspirated at 2, 12, and 24 hours after toxin administration were cultured in the two media and injected into mice. As mice died they were immediately immersed in 5 per cent phenol for 10 minutes; the peritoneum and heart were opened under sterile precautions and the contained liquids were cultured (No.

12, Table 11). Toxin was not present in demonstrable quantities in the dog's blood or peritoneal fluid at any time and was found to disappear from the lumen of the loop between 12 and 24 hours.

The cultures in Experiment 12 showed that it is usually possible to obtain sterile blood from the dog but that the peritoneal fluid aspirated is contaminated with *Staphylococcus albus* and other bacteria. The mice which died after injection of filtered intestinal contents showed the presence in both the heart and blood and peritoneal fluid of staphylococci, streptococci, and *Bacillus coli.*

DISCUSSION

These experiments support the evidence already at hand that an intestinal toxin absorbed directly into the mural circulation is not the agency responsible for deaths from bowel obstruction. No evidence of mesenteric absorption of the toxin injected into the closed loops was obtained. In closed distended intestinal loops, where the mural venous and capillary flow is obstructed through the agency of increased intraluminal pressure, the anoxic effect on the bowel wall results in transperitoneal migration of noxious substances.

CONCLUSIONS

1. *Cl. botulinum* toxin cannot be demonstrated in the blood or peritoneal fluid after its administration by stomach tube to the normal dog, although the toxin can be demonstrated in the intestine. The dog tolerates well *Cl. botulinum* toxin so administered or injected directly into the intestine.

2. Intravenous injection of small amounts of *Cl. botulinum* toxin is lethal for a dog. The blood and peritoneal fluid from such an animal kill mice rapidly.

3. Intraperitoneal injection of *Cl. botulinum* toxin is lethal for a dog, the blood from such an animal being toxic for mice.

4. The presence of *Cl. botulinum* toxin in the blood cannot be demonstrated by the mouse method after injection directly into the dog's normal jejunum through a Biebl loop.

5. *Cl. botulinum* toxin, injected directly into 24-hour closed high jejunal loops exteriorized according to the method of Biebl, could not be demonstrated in the blood or peritoneal fluid of dogs by the mouse method until evidence of seepage through the nonviable bowel wall or perforation of the intestinal segment was present.

6. *Cl. botulinum* toxin could not be demonstrated in the closed loop contents by the mouse method 12 to 24 hours after injection directly into 24-hour closed high jejunal loops of dogs, exteriorized according to the method of Biebl.

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POLYPOID DISEASE OF THE COLON

Anatomical Measurements of the Colon Including Description of a Colonoscope*

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POLYPOID disease of the colon is a pathologic condition that has long been recognized and frequently described. The terminology and classifications used in the literature regarding this disease are confusing. All clinical evidence supports the belief that there is a definite tendency for polypoid lesions of the colon to undergo malignant changes. The etiology of this disease is not understood. However, it appears that multiple polypoid disease limited to a segment of the colon differs etiologically and pathologically from multiple polypoid disease of the entire colon. The diagnosis is suggested by a carefully taken history which discloses changes in the patient's bowel habits, blood in the stool, and indefinite abdominal pain. The use of the sigmoidoscope and double-contrast stereoroentgenographs of the colon establish the presence of polypoid lesions in the colon. Various surgical procedures have been advocated for the early eradication of the tumor tissue. In a recent publication[7] the above statements were discussed in detail.

Recently,[7] I proposed that in selected cases when a segment of colon is involved with polypoid disease or when only a relatively few polyps are present throughout the colon it would seem rational that an attempt should be made to remove the tumors successfully without sacrificing the entire colon. It was proposed that polyps which could not be reached by the sigmoidoscope could be fulgurated through one or more properly placed colostomies.

Anatomical Studies of the Colon and the Description of a Colonoscope

Realizing the danger of perforation of the bowel during fulguration of polyps, an attempt was made to ascertain the thickness of the colon in its various segments. In this study twenty-five adult colons were obtained from the pathology departments of Cook County Hospital and Presbyterian Hospital. From the beginning it was obvious that many sources of error were present. Whether the bowel is found to be distended or collapsed will alter the measurement of its thickness. Microscopic measurements will depend on the method used in cutting the

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sections because it would be impossible to cut each section exactly alike To obviate this, a block of colon measuring 3 by 2½ cm. was removed from each segment, stripped of its fat tags, and weighed. Each section

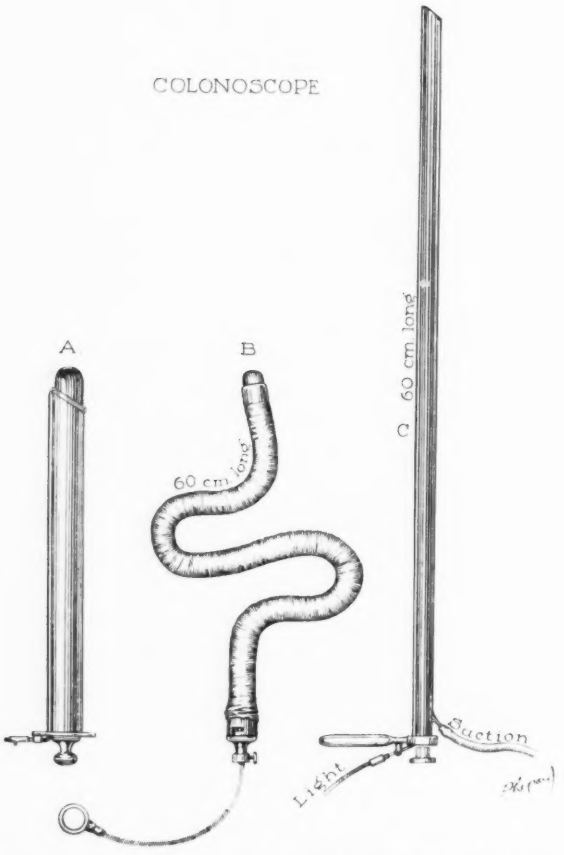

Fig. 1.—Colonoscope, 60 cm. long. *A*, Sigmoidoscope through which a flexible, rubber-covered scope (*B*) is inserted. This flexible portion is used to pass the curves in the lower colon and acts as a sheath for the rigid scope (*C*). It is proposed that the colonoscope will permit fulguration of polyps high in the left colon.

TABLE I

A STUDY OF THE THICKNESS OF TWENTY-FIVE COLONS REMOVED AT AUTOPSY

	AVERAGE WEIGHT IN GRAMS OF PORTION OF COLON MEASURING 3 BY 2½ CM.	TRANSMISSION OF LIGHT, IN FOOT CANDLES, AS MEASURED BY PHOTOELECTRIC CELL*
Rectum	2.139	39.17
Sigmoid	1.888	63.64
Descending	1.437	91.40
Splenic flexure	1.260	92.92
Transverse	1.291	93.68
Hepatic flexure	1.139	98.88
Ascending	1.214	90.28
Cecum	1.166	94.96

*Photoelectric cell readings were made by transmitting the illumination from 100 watt lamp through the tissue from a distance of 1 cm.

was then separately transilluminated and a photoelectric cell was used to measure the amount of light which was transmitted. In this way two methods were utilized in determining the thickness of the colon. The results are tabulated in Table I. It will be seen that the large bowel increases in thickness as it reaches the distal portion, the right colon, thus, being the thinnest and the left colon the thickest. These findings are consistent with physiologic reports[1, 4, 5] which state that the contents of the right colon are liquid and that in this region absorption takes place. The left colon serves as a reservoir[6] for semisolid mate-

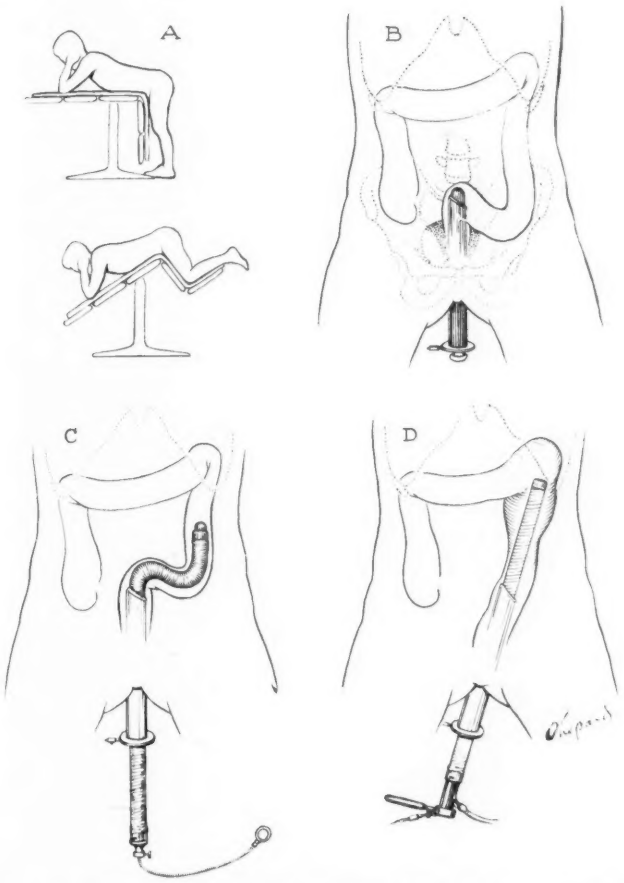

Fig. 2.—Diagrammatic drawings showing method by which colonoscope is introduced into left colon. *A*, Position of patient on table for introduction of scope; *B*, sigmoidoscope in place; *C*, flexible, rubber-covered scope passing around curves in the colon; *D*, flexible tube being straightened out by rigid scope.

rial. This accounts for its greater thickness. It would seem from this study that polyps could be fulgurated in any segment of the colon, but the greatest risk of perforation would be on the right side.

Fulguration of polyps through a scope, passed from below, is a common surgical procedure. This is usually done through 24 and 30 cm.

scopes. Buie[3] on several occasions has introduced a 45 cm. scope and by careful manipulation has been able to fulgurate polyps. However, when these heights are reached, much difficulty is encountered in maneuvering the scope around the curves in the colon.

Colonoscope (Fig. 1).—It occurred to me that by introducing a flexible metal tube through a sigmoidoscope (Fig. 2 *B*) it would be possible to pass the many curves in the lower bowel and reach the upper limits of the left colon (Fig. 2 *C*). When this level is reached, the flexible tube acting as a sheath could be straightened out by a rigid

Fig. 3.—X-ray showing colonoscope inserted well up in the left colon. Note the end of the scope is opposite the twelfth rib.

scope (Fig. 2 *D*). Through this scope, polyps could be fulgurated. The principle on which the colonoscope is based is that a redundant colon, by necessity, has a long mesentery. Such a mesentery will permit the colon to be straightened out without stress, thereby being less painful to the patient.

The flexible portion of the scope is sufficiently pliable to pass any curve in the colon. It is covered with Penrose rubber tubing to prevent traumatizing the bowel. The length of the scope is 60 cm., which is

sufficient to reach the splenic flexure in the average colon. During the above-mentioned studies on the colon, the average distance from the splenic flexure to the anal outlet was found to be 60.4 cm., the longest being 92 cm. and the shortest 40 cm. Results of this study are tabulated in Table II.

TABLE II

LENGTH, MEASURED IN CM., OF VARIOUS SEGMENTS OF TWENTY-FIVE COLONS OBTAINED AT AUTOPSY

	AVERAGE	LONGEST	SHORTEST
Cecum and ascending	28.3	50	19
Hepatic flexure	10.0	12	6
Transverse	28.5	45	17
Splenic flexure	9.9	17	6
Descending, sigmoid, and rectum	60.4	92	40
Entire colon	137.1	192	95

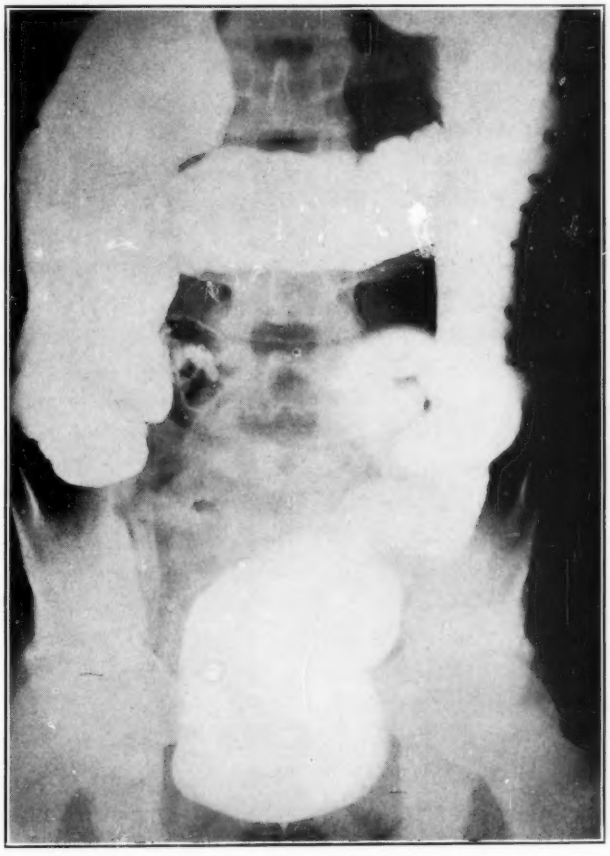

Fig. 4.—Barium enema showing normal colon pattern of patient in whom colonoscope was passed as shown in Fig. 3.

In order to introduce the instrument, the patient is placed face downwards on a tilt table[2] with the head lowered (Fig. 2 *A*). This enables the abdominal contents to fall forward.

That it is possible to introduce this colonoscope is attested by the accompanying x-ray picture (Fig. 3). It shows the scope extending well up in the left colon. Following this procedure, this patient was given a barium enema to show the normal colon pattern (Fig. 4).

It should be appreciated that the introduction of the instrument into the left colon is exceedingly dangerous and should be done only by a trained endoscopist. Also, there are many colons attached by short mesenteries in which it would be impossible to use this instrument.

It is my opinion that further work with this instrument will prove that its use will preclude, in many instances, the necessity for more radical surgery.

I wish to thank Dr. J. P. Nesselrod for his interest and cooperation in the development of this instrument.

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ABSENCE OF THE ANTERIOR MEDIASTINUM WITH REPORT OF CASE ASSOCIATED WITH CONGENITAL DIAPHRAGMATIC HERNIA

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ALTHOUGH compatible with life, an interpleural communication is one of the rarest anomalies of the chest. This is demonstrated by the fact that Kupfer[1] did not mention its occurrence in a study of mediastinal lesions in 6,000 autopsies. Moreover, in a thorough search of the literature only nine cases could be found. One of the earliest cases was recorded by Fräntzel,[2] in 1877, who observed at autopsy bilateral pneumothorax as a result of tuberculosis of the left lung and mediastinum with perforation of the latter structure. McCallum,[3] in 1919, reported another case in a patient with tuberculosis of the right lung. The patient was receiving artificial pneumothorax therapy on the right side, and, after the eighth treatment, fluoroscopy revealed an area of transparency above the diaphragm on the left side. After the ninth treatment, a distinct pneumothorax was seen on both sides. A somewhat similar case was reported by Walsh,[4] in 1924. In 1925, Dumarest and Bonafé[5] reported a case in a man 35 years of age with tuberculosis of the left lung. Within a few moments after the performance of a pneumothorax on the left side, the patient complained of dyspnea and pain on the opposite side of the chest. An immediate roentgenogram revealed air in both pleural cavities, and relief of manifestations was obtained by aspiration of air from both pleural cavities. The case reported by LeWald,[6] in 1926, had no evidence of tuberculosis. The patient experienced sudden pain in the right side while "vigorously" using an adding machine. Roentgenographic examination revealed a right pneumothorax, and one week later there was, in addition to an increase in the collapse of the right lung, a collapse of the left lung. Julien,[7] in 1930, recorded another in a 24-year-old female with pulmonary tuberculosis. During the performance of a pneumothorax, it was observed that, in spite of the fact that air was being introduced into the right side, the left lung became collapsed but the right did not. For unexplained reasons the author concluded that an interpleural communication existed which permitted air to enter the left pleural cavity but not in a reverse direction. A somewhat similar case is cited by Ameuille[8] in which lipiodol introduced in the right pleural cavity was seen to pass into the left through an opening anterior to the heart. Bilateral pneumothorax

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following the production of unilateral artificial pneumothorax was observed in a case reported by Ioganson and Prozorov[9] in 1932. For this reason a mediastinal communication was suspected but not proved. In 1933, Smith and Willis[10] described another in a patient with pulmonary tuberculosis. By means of needles introduced in both pleural cavities it was possible to demonstrate increase in interpleural pressures on both sides during the injection of air on one side. The authors[10] explained the fact that no channel of communication existed at autopsy because of the presence of adhesions between the mediastinal structures and the left lung and the complete obliteration of the left pleural space by adhesions.

In order for such interpleural defects to exist, the mediastinum, bounded by the spinal column behind, the sternum anteriorly, and the pleurae laterally, must be involved. Nitsch[11] has directed attention to two points at which the mediastinum is anatomically weak; viz., the area posterior to the upper part of the sternum in the region of the second to the fourth sternocostal junctions, and posteroinferiorly at the level of the eighth rib. An additional area of weakness between the vertebrae posteriorly and the esophagus anteriorly from the third to the fifth thoracic vertebrae has been described by Maier.[12] The vena azygos separates the upper from the lower posterior weak areas.

The anterior interpleural space contains little in the nature of resisting structure, being filled mostly by fatty and loose areolar tissue replacing the atrophied thymus gland. Korol[13] states that the anterior mediastinal septum may bulge to reach the opposite axilla, allowing the upper lobe of the invading lung to occupy the entire anterior aspect of the opposite hemithorax. In some cases the upper anterior weak spot is represented by only a thin membrane, formed by a fusion of the right and left pleurae. The esophagus is the only organ running the length of the posterior mediastinum with no branches to other structures; therefore, it is subject to displacement. This occurs most readily in its lower part, where it turns forward from the aorta to reach the stomach. Lung hernias occur here between the esophagus and the aorta.

The pleural cavity on either side is protected against contralateral disturbances by the mediastinum. Respiratory movements, which cause a changing diameter of the thorax, make it necessary that this partition be somewhat flexible; however, it is unyielding to such an extent that one side of the thorax is protected from any change in pressure which might occur in the other, and a compensatory function is made possible. According to Richter,[14] the mediastinum insures an equable circulation of blood and guards vessels and nerves from injuries likely to arise from unequal functions of respiration, deglutition, and heart action.

When pressure on either side is increased excessively, the mediastinum may be made to "give," which results from gradual loosening of its fibers. Such deviation of the mediastinum is common in thoracic dis-

case, but it should be differentiated from herniation and interpleural communication. Herniation of the mediastinum is the protrusion of part of the pleural sac contents with evagination of the mediastinal pleura into the contralateral hemithorax. This is common during the administration of a pneumothorax, but, according to Maier,[12] it may result from lesions within the chest even though pneumothorax does not exist. Pulmonary tuberculosis is the most common cause, due to atelectasis and fibrosis. Among other initiating factors are atelectasis from bronchial tumors, extensive unilateral suppurative bronchiectasis with pulmonary contractions and fibrosis, occluding intratracheal foreign bodies, and cystic disease of one lung. Usually the pressure differences have existed for a considerable period of time before herniation occurs.

Korol[13] finds anterior mediastinal hernias as common on the right side as on the left, but he adds that posterior mediastinal bulging is rare on the right. This is explained by the fact that the esophagus relaxes in leftward displacement, but, on being pushed to the right, it resists by stretching, due to its diaphragmatic attachment on the left side. It is easier for the right lung to invaginate itself between the aorta and the esophagus because of the difference in the relations of the mediastinal pleura. The right pleura extends posterior to the esophagus, forming a recess behind it from the tracheal bifurcation to the diaphragm. The left pleura is not in direct contact with the esophagus. In lung prolapse there is no hernial sac, the parietal pleura being ruptured.

The case outlined herewith is one of a communication between the pleural cavities observed during left transthoracic diaphragmatic herniorrhaphy. In contradistinction to the above-mentioned cases, our case is characterized by total absence of any evidence of tuberculosis. In this patient the entire anterior mediastinum was absent, affording a clear view into the right hemithorax for the full length of the sternum. The heart and great vessels lay on the anterior surfaces of the vertebral bodies, and the right lung was compressed to such an extent that it was thought to be congenitally absent.

CASE REPORT

The patient, a white male, age 4 months, was admitted to the Hutchinson Memorial Clinic in July, 1937. The complaint registered by the parents was "grunting, wheezing, and difficult breathing." The present illness had existed for two months. There was occasional cyanosis. The child had a poor appetite and had failed to gain weight since the onset of his trouble. He had been very fretful during the previous two months and had suffered from a hacking cough for the two weeks prior to his admission. Physical examination revealed an undernourished child with prominent frontal bosses, an enlarged head, and bulging eyes. The thorax was large and rounded, convex anteriorly, and breathing was labored; the short, rapid respiratory efforts being produced with marked abdominal effort. The percussion note was dull over the lower half of the left lung posteriorly, tympanitic

over the lower two-thirds of the left lung anteriorly, and hyper-resonant over the entire right lung. The heart and mediastinum were displaced to the right. Breath sounds were diminished over the lower two-thirds of the left lung. The liver was palpable below the right costal margin. Roentgenographic examination of the thorax showed marked displacement of the heart and other mediastinal structures toward the right. There was increased density at the left base and in the hemithorax were scattered air pockets without fluid levels which were apparently gas-filled small and large bowel (Fig. 1). That there were loops of small bowel present in the left hemithorax was demonstrated by roentgenograms made following the ingestion of a barium meal (Fig. 2).

Fig. 1.—Anteroposterior roentgenogram of thorax before operation showing marked displacement of heart and other mediastinal structures toward the right. Increased density in left hemithorax with scattered air pockets without fluid levels suggests gas-filled small and large bowel.

A diagnosis of congenital left-sided diaphragmatic hernia was made. It was considered best not to operate immediately, but to wait until the child was older unless the symptoms became more pronounced, and this course was endorsed by the Department of Pediatrics.

On Sept. 17, 1938, the patient, now 18 months old, returned to the clinic. Breathing had improved and nutrition was satisfactory. Fluoroscopic and roentgenologic studies were done. Retrograde filling of the colon by means of a barium enema showed the sigmoid loop to be fairly straight. This and the descending colon ran upward along the left lateral abdominal wall to enter the thorax through an opening in the diaphragm (Fig. 3). What probably corresponds to the splenic flexure was observed along the lateral chest wall in its middle third at approxi-

mately the level of the seventh rib. The transverse colon passed medially from this point toward the mediastinum. The position of the cecum could not be determined, but at least one loop of intestine, which was probably either cecum or terminal small intestine, crossed the midline in the upper portion of the thorax to enter the right half of the thoracic cage. (Fig. 3.)

Fig. 2.—Anteroposterior roentgenogram of abdomen and chest made four hours following ingestion of barium meal. Loops of barium-filled small bowel may be readily seen in the left hemithorax.

On Sept. 19, 1938, the patient was admitted to the Tulane University Surgical Unit at Touro Infirmary for a diaphragmatic hernioplasty, which was done on Sept. 21, 1938. Under general anesthesia, subperiosteal resection of the seventh left rib was done and the pleura opened in the rib bed. Inspection revealed both small and large intestines herniating without a sac through a defect approximately 3 by 6 cm. in the left diaphragm. A Meckel's diverticulum was found on the herniated terminal ileum, but it was not removed. There was no anterior mediastinum present, the right hemithorax being clearly visible for the entire extent of the sternum. By positive pressure manipulations, the anesthetist could inflate the right lung to fill the pleural space on that side, and at this time it was believed that no left lung existed. On retrospect it is thought that the lung in-

flated was the left and that the right was not observed because it was compressed behind the left. In order to reduce the abdominal viscera, it was necessary to enlarge the incision downward approximately 4 cm., with resection of 2 cm. to the eighth and ninth costal cartilages. The intestines were then returned to the abdominal cavity, and the defect closed with interrupted sutures of No. 1 white silk. The primary suture line was then reinforced with interrupted mattress sutures of the same material. The thorax was closed in layers, interrupted sutures of No. 1 white silk being used throughout. The skin was approximated with

Fig. 3.—Anteroposterior roentgenogram of abdomen and chest made following barium enema. Parts of the transverse colon, splenic flexure, and descending colon may be visualized in the thorax.

interrupted vertical mattress sutures of the same material. A marine sponge dressing was applied, and the patient left the operating room in good condition. Two hours following the operation, a transfusion of 150 c.c. of unmodified blood was given.

The postoperative course was uneventful. On Oct. 3, 1938, the day of the patient's discharge, roentgenologic studies were made. Examination of the chest

showed a restoration of the left diaphragm. The instillation of iodized oil into the tracheobronchial tree revealed both lungs present (Fig. 4). There was very little displacement of the mediastinal shadow and approximately 50 per cent expansion of the left lung had occurred, there being some residual pneumothorax. Examination of the gastrointestinal tract by successive films taken at hourly intervals showed the stomach and duodenum to be approximately normal in position (Figs. 5 and 6). The small gut coils occupied a somewhat irregular position, but function appeared to be quite within normal limits, the stomach, duodenum, and most

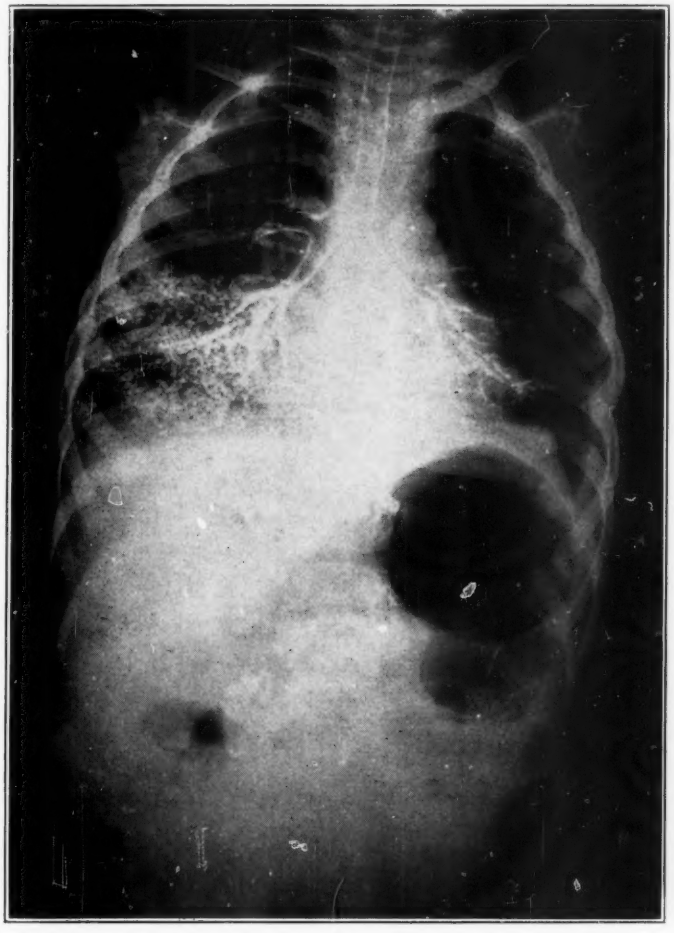

Fig. 4.—Anteroposterior roentgenogram of thorax following instillation of iodized oil into tracheobronchial tree approximately twelve days after operation. Trachea, both main bronchi, and their secondary divisions are clearly visible. There is little or no displacement of the mediastinal structure, but there is some residual pneumothorax.

of the small intestine being empty at four hours with the meal occupying the cecum, which was in the right lower quadrant. At twenty-four hours the barium was seen to be in the descending colon, sigmoid, and rectum, which were approximately normal in position.

On Dec. 10, 1938, about two months after operation, examination of the patient revealed that the thoracotomy wound had healed completely and the lungs and heart were clinically normal. Roentgenologic examination at this time showed the heart and aorta about normal in size, shape, and position. The diaphragms were

smooth and rounded and in normal position. There was evidence of bone regeneration in the region of the left seventh rib. The lung fields were apparently normal. A large gas bubble was observed in the cardiac portion of the stomach (Fig. 7).

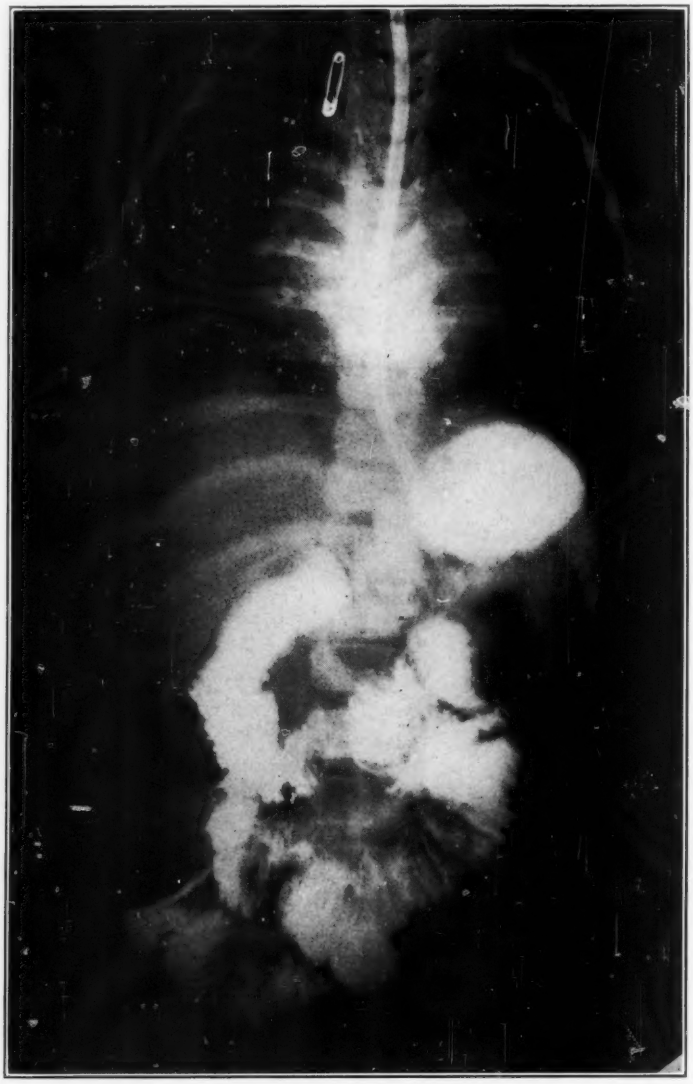

Fig. 5.—Anteroposterior roentgenogram of abdomen one hour following instillation of barium meal in stomach about twelve days after operation. The stomach, duodenum, and the remainder of the small bowel are approximately normal in position.

Whereas the previously described cases[1-9] of interpleural communication were probably due to small defects in the mediastinum with pathologic backgrounds, usually tuberculous, our case is one of a *complete absence of the anterior mediastinum which is undoubtedly of congenital origin.* Moreover, as pointed out by Oechsli and Miles,[15] in some of these cases the communication was suspected only. In these cases, especially those with tuberculous lesions, the possibility of a spontaneous

pneumothorax in the contralateral side must be considered. Our case is unique in that it is the only one which we have been able to find in which the defect in the anterior mediastinum was so great that in reality a single pleural cavity existed. It also differs from the reported cases in that it was definitely congenital. Dr. Harold Cummins, Professor of

Fig. 6.—Lateral view of abdomen one hour following instillation of barium meal in stomach about twelve days postoperatively.

Anatomy at Tulane University, has kindly consented to present the following explanation for this unusual anomaly:

"*Development of the Mediastinal Fault.*—It will be recalled that the four definitively independent body cavities were in communication with each other during the early period of their embryonic development. In an embryo of about five weeks the celom is still one continuous cavity,

though its form and relations indicate clearly which regions are to be isolated as the two pleural cavities and the peritoneal and pericardial cavities. The pleural cavities are represented by passages lying dorsal to the heart, one on either side of the primitive mesenteries of the esophagus and heart. These pleural passages, being very small in comparison, appear as constricted canals uniting the expanded pericardial and peritoneal regions of the celom. At about eight weeks the cephalic

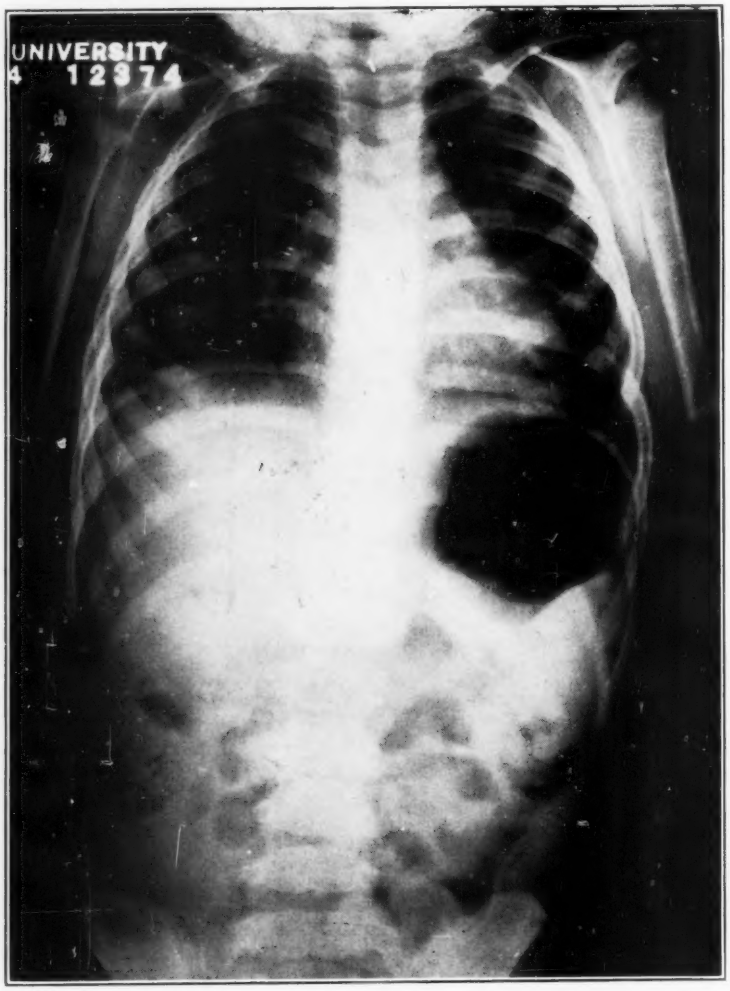

Fig. 7.—Anteroposterior roentgenogram of thorax about two months after operation. Heart and aorta approximately normal in size, shape, and position. The diaphragms are smooth, rounded, and in normal position. The lung fields are normal and there is some evidence of bone regeneration in region of left seventh rib.

and caudal openings of the pleural passages have been completely partitioned from the pericardial and peritoneal cavities. In the meantime the lung buds, evaginating from the medial walls of the pleural passages, continue to enlarge. Accompanying growth of the lung buds, the now enclosed pleural cavities expand. From the original confinement

to a position in dorsal relation to the heart, continued expansion of the pleural cavities extends to the parietal pleurae (and other mesoderm dragged with them) farther and farther around the pericardium, until finally in the fetus near term they nearly meet in front. After birth their relationship is brought even closer in the now more thinned anterior mediastinum.

"Keeping in mind that the anterior mediastinum represents the midline partition persisting after expansion of the parietal pleurae ceases, the history of the mediastinal defect in this case may be reconstructed. There are two possibilities to be considered. (1) The anterior mediastinum may have been initially normal, its absence being due to secondary atrophy under compression by the herniated intestine. (2) The anterior mediastinum may have been obliterated as a result of failure to arrest the progress of expansion of the pleurae toward the midline. Instability of other developmental processes in the present case is indicated by the diaphragmatic fault, and it is not impossible that the behavior of the enlarging pleurae may have been abnormal as well, independently of any direct compressing effects of the herniated intestine. Since, in the dog, the normal history of the pleural expansion is an interruption of the process at a point just short of dissolution of the anterior mediastinum, it is not improbable that stable development, whether in a dog or human fetus, might lead to complete disappearance of the mediastinal septum."

SUMMARY

1. A thorough search of the literature has revealed only nine cases of interpleural communication. The majority of these have been due to small defects in the mediastinum resulting from tuberculous involvement.

2. A case of complete absence of the anterior mediastinum associated with congenital diaphragmatic hernia is presented. This case is unique in that it is the only one we have been able to find in which the defect in the anterior mediastinum was so great that actually a single pleural cavity existed. Moreover, it differs from the reported cases in that it was definitely congenital.

3. The developmental explanation of this mediastinal fault is presented.

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SPLENOSIS: MULTIPLE PERITONEAL SPLENIC IMPLANTS FOLLOWING ABDOMINAL INJURY

A Report of a Case and Review of the Literature

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THE incidence of autotransplantation of splenic tissue following injuries to the upper abdomen is very rare. A case which recently came to our attention has stimulated a survey of this condition. To our knowledge no mention of such a clinical entity has been made in the American literature and we have been able to collect only seven cases in the foreign literature with definite history of trauma and two cases of questionable etiology.

In 1896, Albrecht[1] first noted the presence of multiple splenic nodules throughout the peritoneal cavity. He called these multiple accessory spleens. Schilling, in 1907,[2] found similar nodules on autopsy and also believed they were accessory spleens. In 1910, von Kuttner[3] first brought to our attention the presence of multiple splenic nodules which he found in a patient four years following a gunshot wound of the abdomen that resulted in a rupture of the spleen. Foltin,[4] in 1911, and von Stubenrauch,[5] in 1912, reported a similar condition following severe trauma to the left upper abdomen. Von Stubenrauch in a subsequent paper quoted another case of multiple nodules of splenic tissue found in the peritoneum following an injury to the left upper abdomen. Lee, in 1923,[7] Kupperman, in 1936,[8] and Shaw and Shafi, in 1937,[9] reported similar cases. Each of these cases, with the exception of the first two, gave a definite history of injury to the region of the spleen.

CASE HISTORY

Patient was a 28-year-old Puerto Rican housewife, para ii, gravida iv, who entered Knickerbocker Hospital on Nov. 21, 1938, for treatment of backache of three years' duration and leucorrhea of five months' duration. Family history is noncontributory.

Past History.—Medical: Reveals usual childhood diseases. *Surgical:* In 1919 at age of 9 years, she was in another hospital for ten months after having been struck by an automobile. At this time she had an exploratory laparotomy and was told that her spleen had been removed. *Menstrual:* Menstruation began at 12 years of age and was always regular, occurring every twenty-eight days and lasting four to five days. Dysmenorrhea was always present during the first two days.

In 1936 she first came to the Knickerbocker Hospital Gynecological Clinic complaining of backache. A third degree retroverted uterus was found and temporarily treated satisfactorily with a pessary. She returned in November, 1937, and com-

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plained of recurrent lower back pain and weakness. The uterus was retroverted and tender on motion. A diagnosis of chronic parametritis was made and the patient was given a series of intradermal sterile milk injections with no improvement of her back pain. In April, 1938, postexertional pain developed in both lower quadrants. At this time she also had a leucorrhea. An erosion of cervix was found. Her pain and leucorrhea continued until admission.

Examination on admission revealed a well-developed, well-nourished Puerto Rican female who did not appear acutely or chronically ill. Temperature, 99°; pulse, 98; respiration, 20. Except for abdominal and vaginal findings, her physical examination was negative. There was a firm 3½ inch left upper paramedian scar about 1¾ inches wide. There was very slight tenderness to deep palpation in both lower quadrants. No abdominal masses could be felt. Vaginal examination revealed a firm and smooth cervix which was in the axis of the vagina and had a transverse laceration. There were fullness and tenderness in right fornix and the adnexae were somewhat thickened. The body of the uterus was not enlarged; it was anterior in position and pulled to the right. *Laboratory Data:* Urinary findings were normal. Blood count revealed a moderate anemia. Cervical smear showed a few white blood cells but no intracellular organisms. On her sixth hospital day, a laparotomy was performed for chronic adnexitis and cystoma of right ovary.

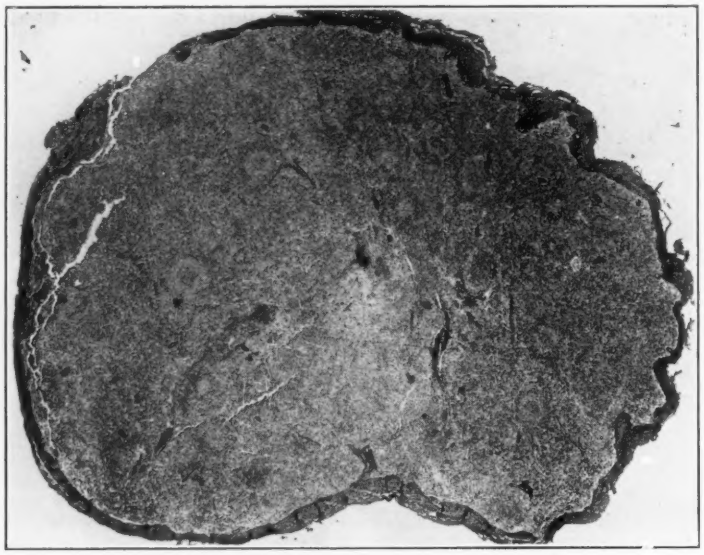

Fig. 1.—General topography of nodules (×15).

Operation.—The peritoneal cavity was opened and the pelvis explored through a 3-inch Pfannenstiel incision. The uterus was pear-shaped, smooth in surface and contour, was situated anterior, firm, and not enlarged. The Fallopian tubes were moderately injected but not indurated. The walls were thin and the lumina patent. The ovaries contained numerous follicular cysts, the right was larger than the left and contained a hemorrhagic cyst and several corpora lutea. On the parietal peritoneum, greater omentum, ileum, ascending colon, and sigmoid were numerous purple-red, sessile nodules measuring 1 mm. to 2 cm. in diameter. These nodules were covered with peritoneum and were intimately connected to the structures to which they were attached. These tumors were soft to touch and grossly resembled hemorrhagic cysts. The surface of each nodule was smooth and its contour was

regular. The masses were situated near the mesenteric attachment of the intestine as well as on its free border. The tissue surrounding each nodule showed no evidence of any gross anatomic change.

The viscera of the upper abdomen could be neither palpated nor visualized because of dense adhesions. The splenic site was entirely covered with thick bands which prevented the determination of the presence of any splenic tissue. We felt at this time that we were dealing with a case of diffuse endometriomas and decided to remove both ovaries. Nodules were removed from ileum, sigmoid, and omentum for pathologic study. The abdomen was closed in layers with interrupted absorbable sutures.

The postoperative course was complicated by moderate abdominal distention and occasional vomiting. Temperature remained normal. Wound healed by primary intention and patient was discharged on the eleventh postoperative day.

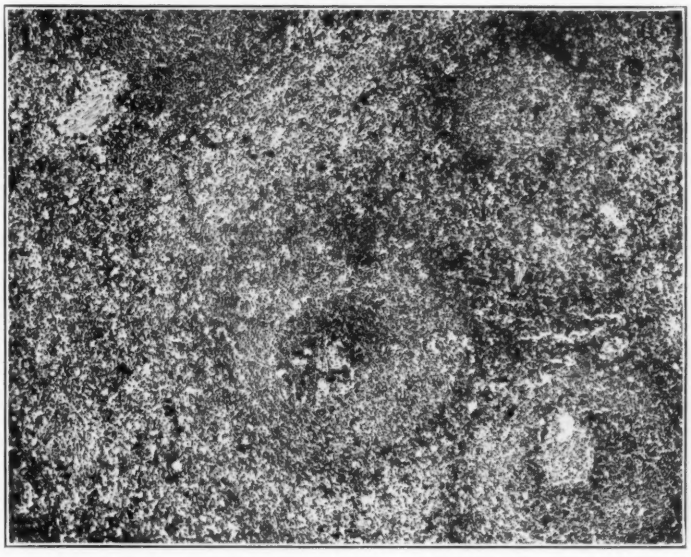

Fig. 2.—Trabeculae and follicle formation. Clear spaces indicate sinuses. (×70.)

Pathology Report.—Upon microscopic examination of the nodules, they presented a collagenous capsule containing a few scattered smooth muscle fibers and a small number of elastic fibers (Fig. 1). Emanating from the capsule were some delicate strands of connective tissue containing elastic fibrils which traversed in broken fashion the peripheral portion of the nodule. The arteries branched for a short distance with these trabeculae, but were subsequently seen deep in the structure of the nodule entirely free of any supporting trabecular-like substance. The background of the nodule consisted of a rich reticular network, the fibers of which communicated with the adjacent trabeculae. The reticulum formed well-defined sinuses within the lumina of which were lymphocytes, occasional mononuclear cells, and polymorphonuclear leucocytes. The intersinusoidal areas contained lymphocytes and large monocytes, consisting of pale vesicular nuclei enclosed in a fairly rich, pale eosinophilic cystoplasm. Several well-defined follicles were seen (Fig. 2), one or two of which contained minute hyaline foci. The histologic characteristics were those of splenic nodules.

DISCUSSION

The problem which immediately presents itself for discussion is whether these nodules were implants or accessory spleens. The usual

location of accessory spleens is in the gastrolienal ligament. However, they have also been found about the pancreas, rarely in the omentum.[13] Their usual number is two to six, but as many as forty have been described.[12] Following splenectomy the accessory splenic tissue has been known to undergo moderate hypertrophy. Although accessory spleens are one of the most commonly found congenital anomalies at autopsy, they are always confined to the locations mentioned above. It is noteworthy that only in those cases in which there was a history of severe upper abdominal trauma were splenic nodules located in such atypical locations as the pleural cavity, the dome of the diaphragm, and on the peritoneal surfaces of the small and large intestines and even of the liver. In view of the history of trauma and the bizarre location of the splenic nodules in our case, we believe it was one of splenic implantation rather than of multiple accessory spleens.

Whether the cases of Albrecht[1] and Schilling[2] were accessory spleens or multiple implants, it is difficult to say. In Albrecht's case[1] there was no history of trauma elicited, but on post mortem many nodules were found in the greater omentum, in Douglas' pouch, and on both domes of the diaphragm. There was severe scar tissue about the left kidney and about the spleen. It is possible for traumatic rupture of the spleen to take place and a patient to recover without operative interference. Schilling's case[2] was that of a 47-year-old female who died of carcinoma of the cervix with invasion of the bladder and a right pyelonephritis. There was no history of trauma in this case either, but there were several nodules found on the peritoneum. On the greater omentum were forty-two nodules ranging from the size of a pea to that of a cherry with microscopic structure of spleen. Lymph follicles were seen in many of the nodules.

It appeared to us that in the following cases there is no doubt that at the time of rupture of the spleen there was a "seeding" of the peritoneum by splenic pulp, rather than a hypertrophy of pre-existing accessory spleens.

CASE HISTORY (VON KUTTNER)

A man was shot in the abdomen during a hunting trip. Under the most primitive conditions, a laparotomy was performed four hours later and the spleen removed. A small piece was left in situ. Four years later he died of coronary sclerosis. An autopsy was done by von Becke. Eighty to one hundred nodules were found scattered throughout the peritoneal cavity. Two nodules, the size of a peach and of a cherry, were found at the site of the spleen. The smaller nodules had microscopic structure of splenic pulp but had no trabeculae or lymph follicles. However, the nodules at the splenic site were typically spleen.

Kuttner[3] believed that the case he reported was one of accessory spleens and not implants. Foltin[4] was the first to suggest that they were splenic implants.

CASE HISTORY (FOLTIN)

A 9-year-old boy received a severe abdominal trauma in a street accident. A short time after the accident a ruptured spleen was removed. During the next six years, the patient had recurrent attacks of pain in right lower quadrants. Laparotomy was performed about six years after the first operation for a chronic appendicitis. Multiple nodules were found throughout the abdominal cavity. One was removed from the sigmoid. On microscopic examination, it showed typical structure of spleen, including lymph follicles.

CASE HISTORY (VON STUBENRAUCH)

A splenectomy was performed on a male who thirty-six hours previously had been crushed between a pole and a cart. The spleen had been torn in two with a 10 to 12 cm. separation. Ten months later the patient was operated upon for ileus. At this time a number of tumors ranging from the size of hemp seed to pea size and with color of spleen were found on the greater omentum, transverse mesocolon, and small intestine. Histologic examination showed a thick fibrous capsule sending trabeculae, unlike splenic trabeculae, into the interior. The pulp was typical of spleen. However, no typical lymph follicles were present but only indefinite collections of lymphocytes. In Oltman's case multiple nodules of splenic tissue were found on the peritoneum subsequent to traumatic rupture of the spleen. Von Stubenrauch concluded that the nodules were formed from "seeds" of splenic pulp scattered throughout the peritoneum at the time of rupture.

CASE HISTORY (LEE)

A 29-year-old male with a history of splenectomy after a crushing injury to the abdomen at the age of 14 years had enjoyed good health until one day before admission when he developed symptoms of ileus. On laparotomy the small intestine was found distended and studded with small, sessile, greenish black tumors. Obstruction was due to an adhesive band from the site of old scar to small intestine. The whole peritoneum covering small intestine and mesentery was covered by sessile and pedunculated tumors varying in size from that of a pinhead to 1½ inches, about 200 to 300 in number. Microscopically, they showed splenic tissue with trabeculae and follicles.

Lee made no comment as to whether these nodules were implants or not.

CASE HISTORY (KUPPERMAN)

A 15-year-old boy was run over by a tram and sustained a ruptured spleen. A splenectomy was done. Six months later a laparotomy was performed in the course of repairing a hernia and diastasis of the wound. About 100 nodules were seen on the small and large intestines. These grossly and microscopically resembled spleen.

Kupperman labeled these nodules "accessory spleens."

CASE HISTORY (SHAW AND SHAFI)

A 20-year-old Egyptian male was admitted in coma and died of uremia. History disclosed that the patient had had a trauma to his left side followed by splenectomy. On autopsy the spleen was found absent. There were no accessory spleens present. Eighty-two transplants, varying in size from 0.2 to 2 cm., were present, eighty on peritoneum, one in left pleural cavity, and one imbedded in

the left margin of the liver. All were covered by serosa. Some were sessile and others pedunculated. One-half were distributed on diaphragm, greater omentum, and pouch of Douglas. Six were on peritoneal surface of the old surgical incision.

The implants at the site of the surgical incision, the authors suggested, may have been implanted there when the spleen was removed through the wound. A rent in the diaphragm accounted for the pleural transplant since there were old dense adhesions between the base of the left lung and the posterior end of the left dome of the diaphragm.

Microscopic examination of nodules showed that blood vessels did not enter through a hilus but branched into smaller vessels which penetrated the capsule around its circumference. The capsule was unlike normal spleen; there was no smooth muscle and elastic tissue was poorly formed and merged with surrounding tissue. Septa were similar to the capsule. The pulp was exactly that of spleen but in only two of the transplants was there an attempt at lymph follicle formation. An additional in-

TABLE I

SUMMARY OF CASES IN THE LITERATURE

AUTHOR	DATE	REASON FOR RELAPAROTOMY	TIME AFTER SPLENECTOMY	LOCATION OF NODULES	NUMBER
Albrecht	1896	Autopsy	No history of trauma or splenectomy	Greater omentum, Douglas' pouch, and both domes of diaphragm	Numerous
Schilling	1907	Autopsy	No history of trauma or splenectomy	Greater omentum, peritoneum of anterior abdominal wall and of pelvis	42 on greater omentum, several more on peritoneum
Von Kuttner	1910	Autopsy	4 yr.	Scattered throughout the peritoneum	80 to 100
Foltin	1911	Chronic appendicitis	6 yr.	On loops of large and small intestines	Numerous
Von Stubenrauch	1912	Ileus	10 mo.	Greater omentum, transverse mesocolon, and small intestine	Numerous
Oltman (quoted by von Stubenrauch)	1919	Not stated	Not stated	Throughout peritoneal cavity	Numerous
Lee	1923	Ileus	15 yr.	Small intestine and mesentery	200 to 300
Kupperman	1936	Postoperative hernia	6 mo.	Small and large intestines	100
Shaw and Shafi	1937	Autopsy	Not stated	Abdominal wall, pleural cavity, liver, diaphragm, Douglas' pouch, and greater omentum	82

teresting microscopic finding was the presence of several miliary tubercles which were found in transplants (patient had fibrous caseous apical tuberculosis).

In this case the pulp was apparently the only tissue of splenic origin in the nodules; the capsule and septa were believed to have been derived from surrounding tissue; the vascular supply entered through periphery rather than through a hilus.

Shaw and Shafi[9] expressed the opinion that these splenic nodules were truly implants and not accessory spleens. They based their opinion on the peculiar location of the nodules, their great number, and their atypical microscopic appearance.

It has been shown experimentally that splenic tissue may be implanted in the peritoneum and readily survives transplantation. Von Stubenrauch[6] removed the spleen in several dogs, crushed the pulp, and "seeded" these throughout the peritoneal cavity. On sacrificing the dogs from one to three months later, he was able to demonstrate the same picture he had described in man. Marine and Manley, in 1920,[10] and Perla, in 1936,[11] showed the readiness with which autoplastic splenic transplants grow in rabbits and albino rats although these transplants were into the abdominal wall and not the peritoneum.

SUMMARY AND CONCLUSIONS

1. A case is reported of autotransplantation of splenic tissue throughout the abdominal cavity following trauma of the spleen.

2. The surgeon should bear the above condition in mind in a patient having a history of severe abdominal trauma, since these nodules may grossly resemble endometriomas.

3. These implants histologically resemble normal splenic tissue except for paucity of lymph follicles, trabeculae, and occasionally in atypical arrangement of blood vessels.

4. The great number, the widespread location unusual for accessory spleens, and the history of severe trauma to the spleen lead us to believe that these nodules are not accessory spleens but rather definite splenic implants.

5. There were no abnormal manifestations from the splenic nodules in spite of their widespread distribution and intimate contact with the abdominal viscera in any of the reported cases.

6. We offer the term splenosis to describe this condition.

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A NEW TECHNIQUE FOR CUTTING SKIN GRAFTS

Including Description of New Instruments*

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THE cutting of satisfactory skin grafts of proper size and thickness is an art mastered by few. If, by any practical means, it could be reduced to a simple technical procedure, the grafting of fresh and healthy granulating areas would be practiced more universally, thus avoiding the formation of many unsightly and disabling scars and effecting a great economic saving to the individual patient and to the community. Also, it would be a great advantage to be able to obtain more usable graft from a given donor area, as well as single grafts of a sufficient size to cover large defects completely when good cosmetic results are imperative.

Satisfactory grafts can be cut if the following conditions are fulfilled: (1) a suitable donor surface, (2) satisfactory instruments, and (3) an operator of sufficient skill in the art of cutting skin grafts. A number of devices[1-4] have been described to facilitate the cutting of skin grafts, but unfortunately none of these schemes are entirely practical. Blair and his coworkers have described a very good suction retractor and knife, but their use requires more than ordinary skill.

It is relatively simple, mechanically, to devise a knife equipped with a guide to cut to an even depth into a uniform donor area. To obtain a sufficiently uniform surface, it is merely necessary to stretch the donor skin moderately taut and elevate it slightly above its surroundings. The required instruments and a practical technique will be presented.

DESCRIPTION OF THE APPARATUS AND TECHNIQUE

A detailed description of an instrument would be inappropriate. However, for an intelligent understanding of its technical use, the fundamental principles of its make-up must be outlined. The complete armamentarium consists of a pair of needle retractors for the skin, a traction bar to maintain uniform skin tension, a holder for the razor blades which serve as the cutting edge, and a frame to carry the cutting edge and regulate the depth of the cut (Fig. 1).

The needle retractors, Fig. 1*A*, are pressed into the skin and attached to the traction bars, Fig. 1*B*, under the desired tension. This gives a flat, uniform surface appropriate for the cutting of a graft (Figs. 5-8).

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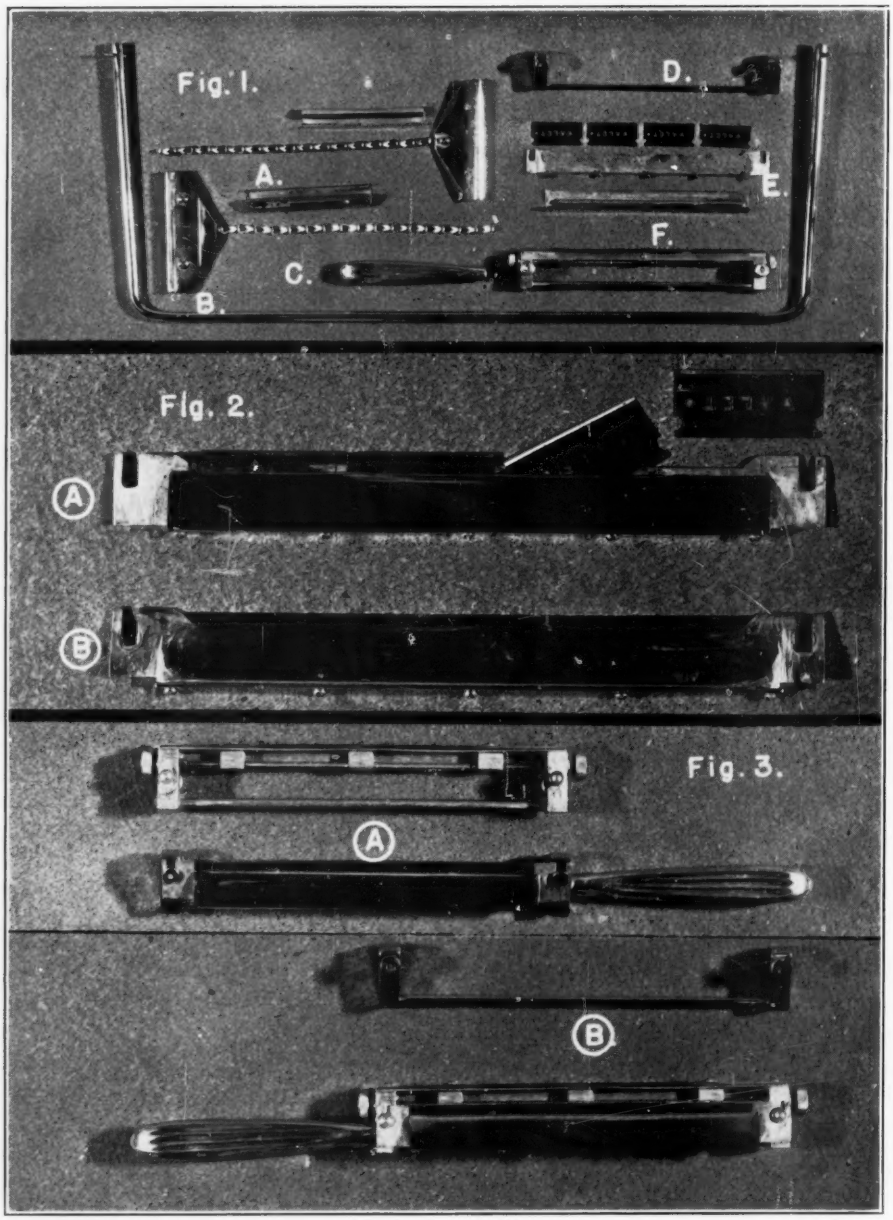


Fig. 1.—The complete armamentarium unassembled to demonstrate the various parts: *A*, two needle retractors; *B*, one traction bar; *C*, a detachable handle, which can be secured into either end of *D* or *F* to make both right- and left-handed instruments; *D*, a simple frame; *E*, a group of four valet autostrop blades, and the holder for the blades which has been taken apart; and *F*, a frame with an adjustable depth guide.

Fig. 2.—Illustrating the blade holder and manner of changing blades. *A*, The blades are slipped into the holder and held rigidly in place by two flat springs. *B*, The assembled holder and blades.

Fig. 3.—*A*, The handle and cutting edge are attached to the simple frame to give a knife for free-hand cutting. *B*, Here the handle and cutting assembly are attached to the frame fitted with an adjustable depth guide. Note: The handle can be attached to either end of the frames for use both as right- and left-handed instruments.

A folded towel is placed under each retractor to elevate the stretched strip of skin slightly above the level of the surface of its surroundings.

The cutting edge consists of four valet autostrop safety razor blades secured end-to-end in a simple holder (Fig. 2). This cutting edge is extremely sharp, since new blades can be used at each operation. These blades are available at any corner drugstore and eliminate the bother and expense of sharpening special knives which present a real problem in other than metropolitan areas. This cutting edge is attached to the frames illustrated in Fig. 3*A* and *B*. The schematic drawing (Fig. 4) illustrates the principles of the assembly.

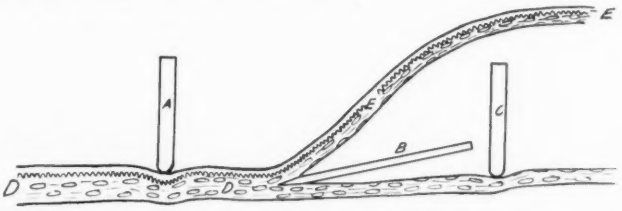


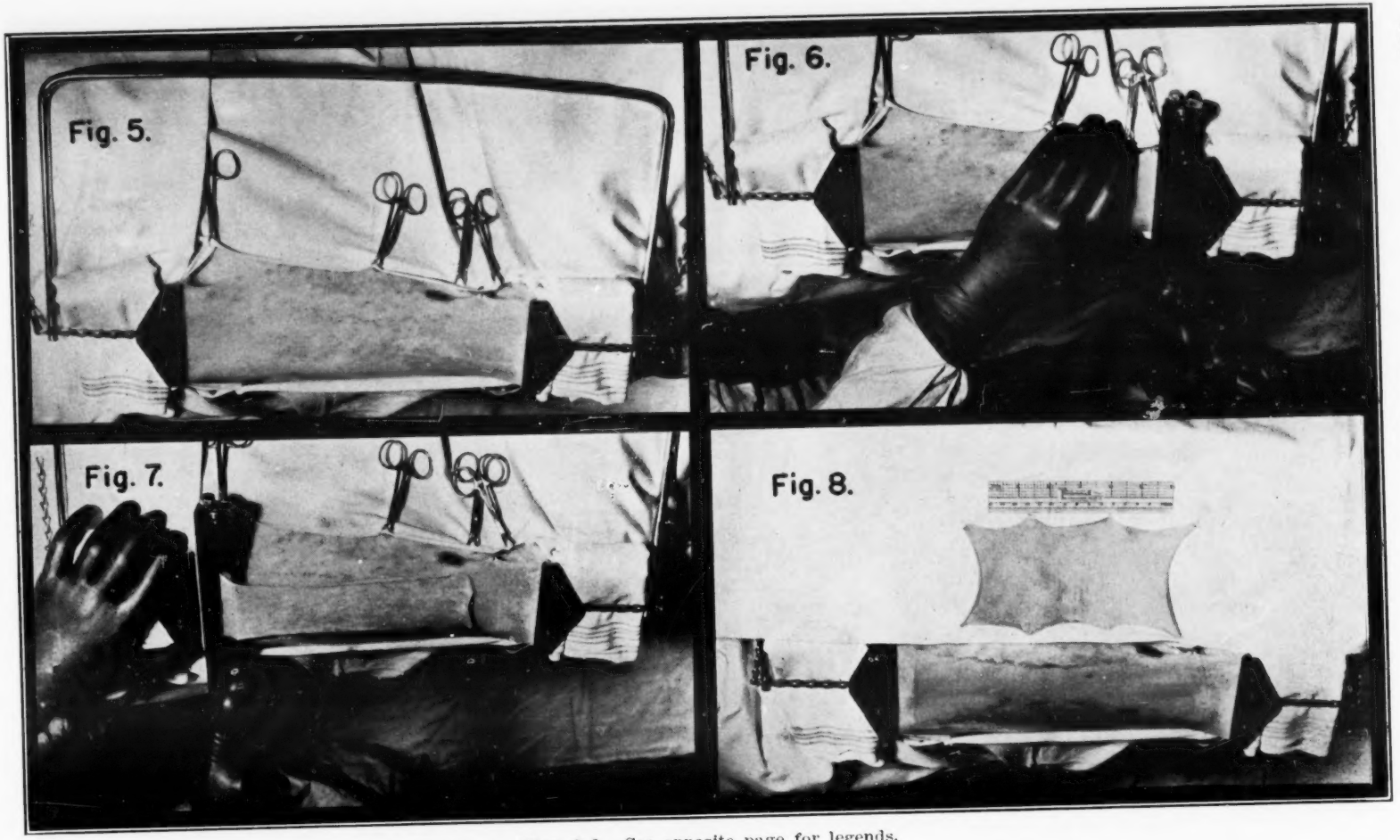
Fig. 4.—Illustrating the principles of the apparatus. The full thickness of the skin, *D*, is stretched taut in practically a plane surface. The depth guide, *A*, rides on the skin surface in front of the blade, *B*. The guide, *C*, rides on the cut surface behind the blade. The blade, *B*, is suspended between *A* and *C*. *A* can be adjusted up and down to cut the desired thickness of graft, *E*. *B* is an extremely sharp razor blade and cuts without pressure. This results in grafts of unusually uniform thickness.

The thickness of the grafts is regulated by adjusting the depth guide *A*, Fig 4. A piece of skin under tension naturally tends to lengthen, and so the graft obtained will be wider and shorter than the path actually cut. The relaxed graft behind the knife is about 20 per cent wider than the path cut on the stretched surface (Fig. 8).

DISCUSSION

This technique is extremely simple and can be acquired by any surgeon of ordinary dexterity. Suitable grafts will be obtained at the first attempt and, with a little practice, large grafts of uniform, desired thickness can be cut routinely. Since the skin being cut is under tension and raised above the surrounding area, the cut edges will be abrupt and more serviceable graft can be obtained from a given donor area. Also, since the skin is placed under tension, it is just as simple to cut grafts from a loose, atrophic skin on any part of the body as from a firm, smooth thigh.

A graft is readily cut to pattern. Obviously, the pattern must be outlined on the donor area before it is placed under tension. The borders are controlled by applying pressure ahead of the blade at the desired points. In this manner tattoo marks are easily excised, provided the pigment does not involve the full thickness of the skin. The healing of the donor area and the use of the grafts are the same as in any other procedure. Large grafts of uniform thickness are routinely obtainable.



Figs. 5-8.—See opposite page for legends.

CONCLUSION

A simple technique and practical armamentarium have been described which, with little practice, will permit the surgeon of ordinary skill to cut excellent grafts of any desired thickness.

The method is rapid. The instruments are sturdy and easily adjusted. A novel cutting edge has been provided which eliminates special knives. The use of a standard safety razor blade has eliminated dependance on an expert knife grinder and has made an unlimited supply of sharp cutting edges available in out-of-the-way places. This should prove to be of considerable advantage and encourage the use of the proper type of skin graft for covering defects at the optimum time.

This method of cutting grafts in no way alters the healing of the donor area or the technique of applying expertly obtained grafts, and so no discussion of this phase of skin grafting is given.

The use of the retractors and traction bar to fix and elevate the donor area facilitates the cutting of grafts with an ordinary knife.

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Fig. 5.—Illustrating the application of the needle retractors and the traction bar to give a donor surface under uniform tension and raised slightly above the surrounding skin.

Fig. 6.—Illustrating the technique of cutting. The left hand and fingers make lateral pressure on the edges of the path to be cut. This maneuver elevates the donor surface and enables one to cut the edges abruptly. At this stage the depth guide is adjusted to give the desired thickness of graft.

Fig. 7.—Illustrating the completion of the cut.

Fig. 8.—Illustrating the graft and the donor bed. The relaxed graft is shorter and wider than the taut donor bed. The degree to which this occurs depends on the character of the donor skin. The edges are sharp and abrupt. Note: Little bleeding occurs at the donor site while tension is maintained. If the tension is continued for a short time, little bleeding will occur subsequent to its release.

A NEW APPENDICAL CLAMP

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THE instrument herein described combines the function of the traditional split sponge used to protect the cecum during the amputation of the appendix and the clamp usually employed to empty the lumen of and anchor the appendix to the split sponge. It is composed of an upper and a lower arm. To the upper arm is attached, by means of a spring, a blade which milks the appendical lumen empty of its contents as it slides over the appendix while closing the clamp. The lower arm is supplied with a flat blade with a slit in it, which takes the place of the split sponge.

Once the mesoappendix is tied and the appendix is freed and ligated, the clamp is introduced by inserting the appendix at the constriction produced by the ligature into the slit in the flat plate on the lower

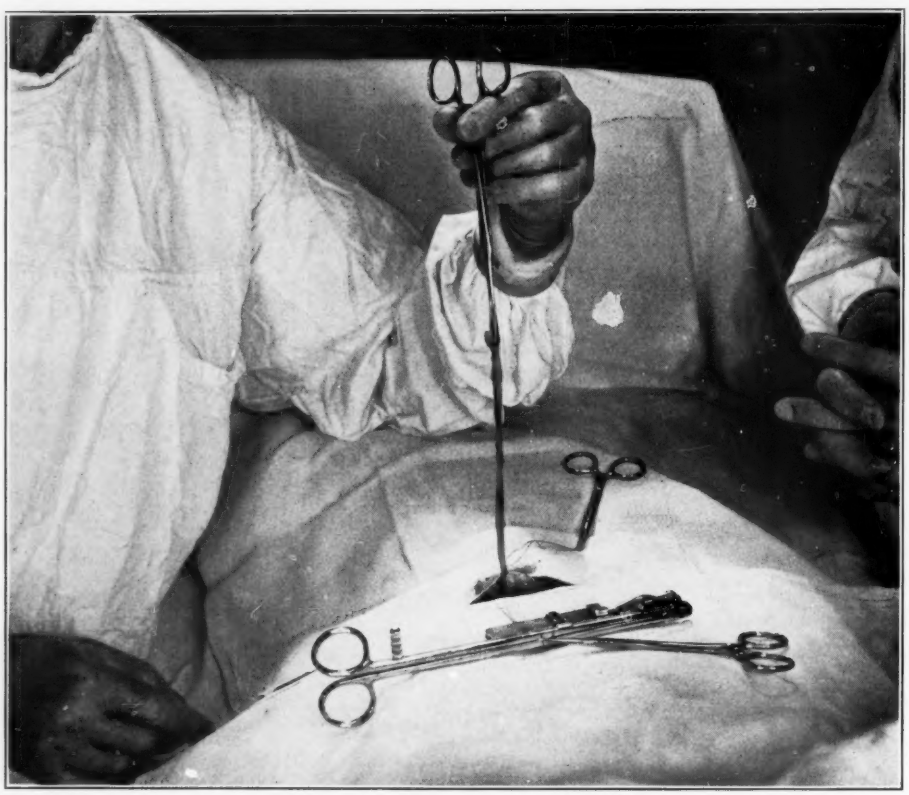

Fig. 1.—Closed clamp by freed appendix.

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arm of the clamp. The organ is then laid flat on the broad plate attached to the lower handle of the clamp. The upper arm of the clamp is brought down while the blade attached to it slides over the base of the appendix from the ligature up, milking thereby the appendix and emptying its lumen at the site of proposed section. The clamp is locked, thus anchoring the appendix firmly to the clamp preparatory to section. The appendix is then severed half-way by the carbolic knife or cautery beyond and between the border of the sliding

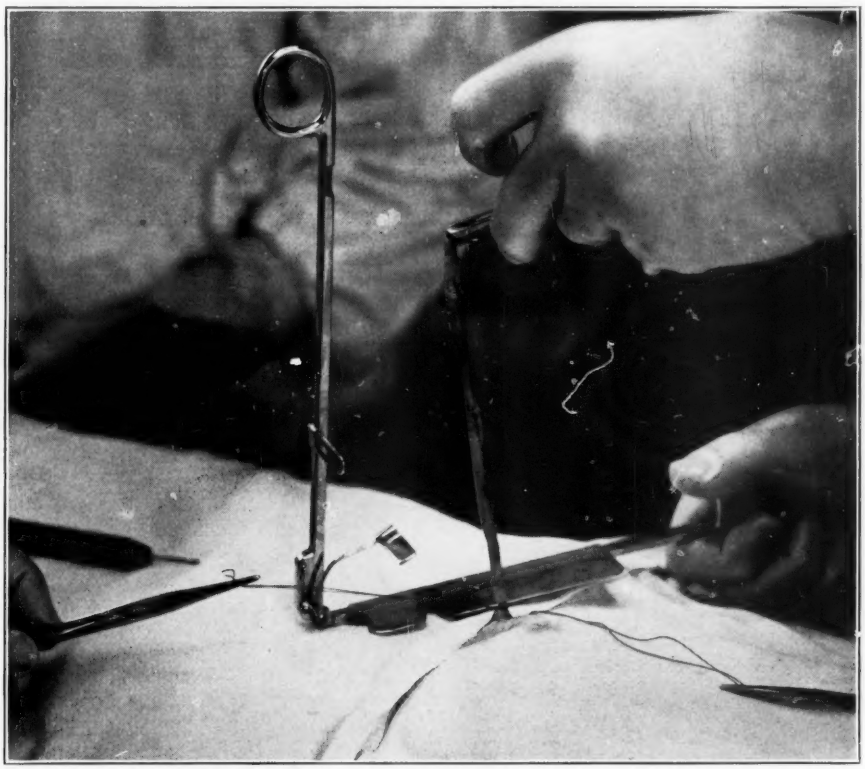

Fig. 2.—Clamp opened preliminary to insertion of appendix.

blade and the ligature. The lumen of the stump is cauterized and the section of the organ is then completed. The clamp is withdrawn and discarded with the appendix still attached. The ligature on the stump is cut and the cecum reposed with or without inversion of the stump.

The use of this clamp facilitates emptying the lumen of the appendix at the site of section; protects the cecum from injury with the knife, contamination with carbolic, or burn with the cautery; and simplifies the discardure of the amputated organ. This clamp has the added advantage over the gauze sponge of not being inflammable when employing the thermocautery.

Fig. 3.

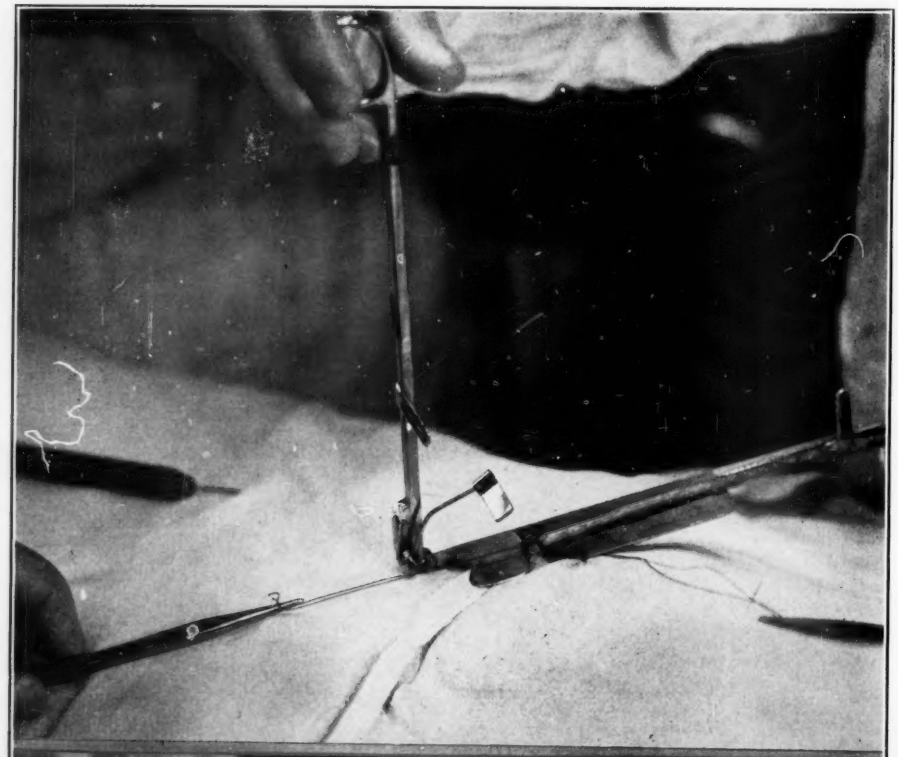

Fig. 4.

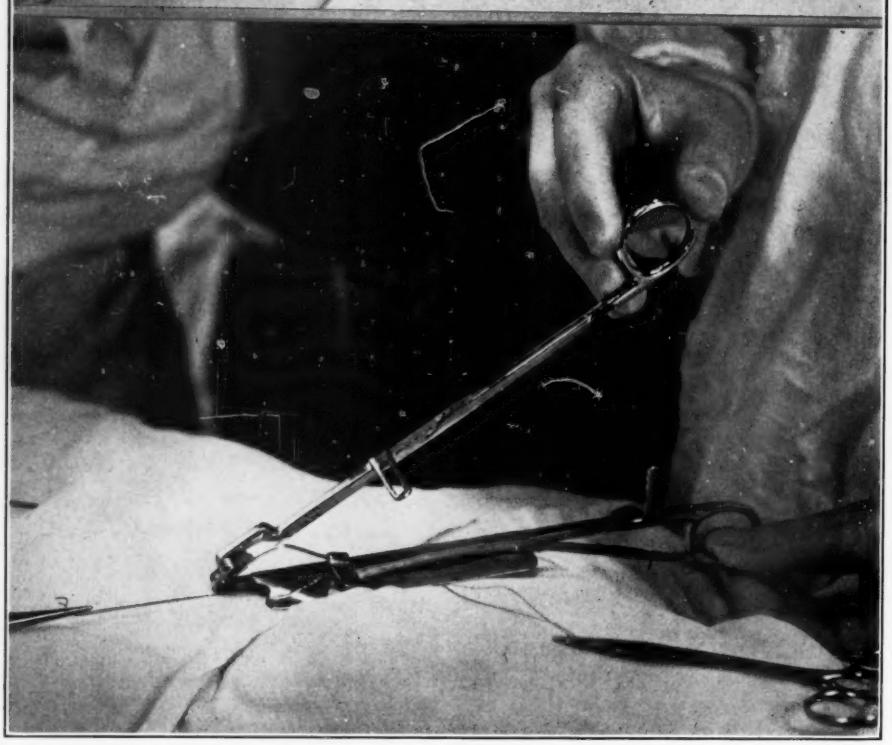

Fig. 3.—Appendix in place.
Fig. 4.—Clamp being closed; sliding blade milks appendix empty.

Fig. 5.

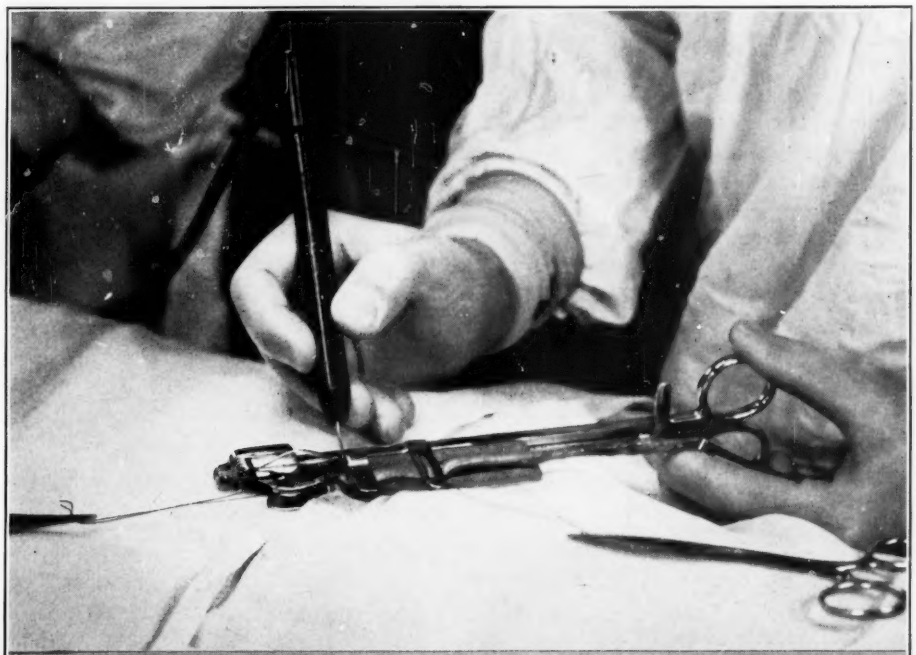

Fig. 6.

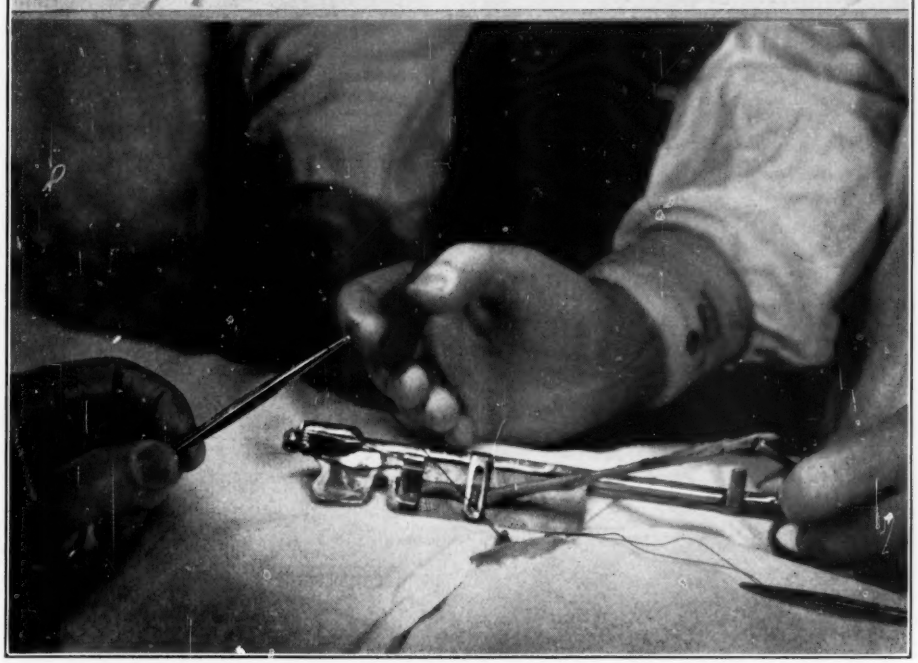

Fig. 5.—Clamp locked and cautery in use.
Fig. 6.—Clamp with appendix attached is withdrawn.

PERIRENAL LIPOMA

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THE case here reported is unusual, both because of the rarity of perirenal lipoma and because of the satisfactory recovery of this patient after removal of the tumor. It is of interest to the surgeon and the pathologist, as few have encountered this tumor at operation or autopsy.

A review of the literature in 1929 by Elansky[1] revealed 150 cases; since his report there have been described fifty-four additional cases.[2-4] Students of the subject have found the immediate postoperative mortality to be about 20 per cent. The immediate cause of these deaths is shock from hemorrhage (spleen, liver, vena cava), injuries, or long operative procedures. Among later complications causing death are frequently listed peritonitis, necrosis of the intestine, phlebitis, pneumonia, and pulmonary embolism. Lacene,[5] in discussing the prognosis, cites an eventual mortality of 54 per cent in the cases that were followed. The high mortality is largely due to late recurrences, many of which proved to be inoperable sarcomas.

CASE REPORT

The patient, a white female, 62 years of age, was admitted to St. Margaret's Hospital, March 13, 1939, complaining of a mass in the left side of her abdomen. She stated that she had been aware of the presence of the mass for the preceding two years, but it had not caused her any marked discomfort other than occasional mild colicky pains in the region of the mass. There was no history of nocturia, hematuria, urgency or frequency of urination. Fearing that she had a cancer, she visited her physician who advised hospitalization.

Physical examination revealed a 62-year-old white female, fairly well nourished and well developed, lying in bed, not acutely ill, in no apparent pain or distress. Blood pressure, 170/64; pulse, 80; respiration, 20; temperature, 98.5° F. The head was essentially negative, and the chest symmetrical. The breasts were large and flabby. The lungs were clear. The abdomen was moderately rounded and palpation revealed no rigidity, increased resistance, or tenderness. In the left upper quadrant was felt a mass about two inches wide and approximately four inches long that could be rolled beneath the examining hand. It was soft and spongy and its edges were sharply defined.

Urinalysis revealed a specific gravity of 1.010, acid in reaction, with a slight trace of albumin, but no sugar or acetone, few white blood cells, many urate crystals. Blood count showed 3,800,000 red blood cells; hemoglobin, 70 per cent; white blood cells, 7,000. The differential count showed 70 per cent polymorphonuclear leucocytes, 24 per cent lymphocytes, 6 per cent monocytes.

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A pyelogram with diodrast revealed, in the preliminary plate, no stone in the genitourinary tract. Diodrast: seven minutes, right kidney was normal in appearance, diodrast was excreted; nonfunction of the left kidney; fifteen minutes, normal function of right kidney, no function of left. There is a large tumor of left abdomen. Preoperative diagnosis is tumor of left kidney.

Operation was performed March 15, 1939, and tumor of left kidney excised. Left lumbar incision extending from the costovertebral angle downward, swinging forward and terminating 1 inch above and 2 inches in front of anterior superior spine, muscle and fascia divided and retracted, exposing the perirenal region. Exploration revealed a large mass extending well up beneath the diaphragm and down in the pelvis. Careful dissection with the hand freed the mass at all points except in the region of the kidney pedicle. Kidney was lying above and in front of this mass. Mass was delivered through the abdominal wound and that portion which refused to separate was caught between two clamps, divided, and ligated. Exploration at this point revealed a large cavity extending up underneath the diaphragm and down in the pelvis and the kidney situated in the upper and anterior portion of this cavity. Nephrorrhaphy was done by the method of Edebohl. Wound was closed in layers with drain down in the lower portion of this large cavity.

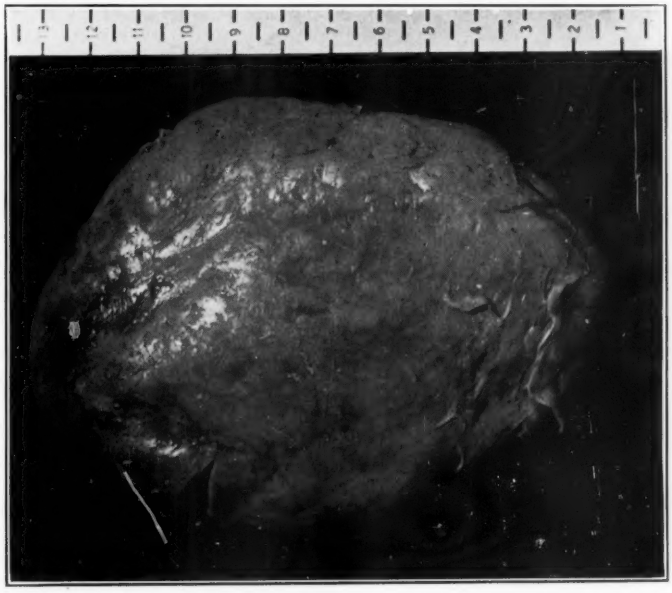

Fig. 1.—A perirenal lipoma weighing 6¾ pounds, successfully removed.

The specimen consisted of a large mass of fatty tissue, measuring 30 by 25 by 8 cm. and weighing 6¾ pounds. The entire tumor was covered with a delicate connective tissue membrane, which could be considered a capsule. On section, it was found to be made of normal appearing fatty tissue with a suggestion of gross lobulation. Microscopic examination of sections taken from various parts of the tumor revealed normal fat cells enclosed in a delicate connective tissue.

The patient had a satisfactory postoperative course and was discharged on March 28, 1939.

DISCUSSION

The diagnosis of perirenal lipoma is rarely made preoperatively. Its rarity and the lack of early and significant symptoms probably account

for this fact. The diagnosis rests on finding a large abdominal tumor, soft and semifluctuant, with little or no mobility, extending into the flank in the region of one or both kidneys and presenting a history of slow growth. A thorough cystoscopic and pyelographic study is helpful in differentiating this tumor from those of kidney, ovary, pancreas, and intestine. However, an accurate diagnosis is not essential as the presence of a tumor with or without symptoms should be sufficient indication to perform exploration.

Various theories have been advanced as to the nature of the pathologic process responsible for lipomas, but the etiology of these tumors is unknown. There is a striking tendency for these tumors to occur in certain locations. In the male they are particularly common around the neck, and in the female they appear most frequently in the breast. They also occur retroperitoneally, and in this latter group the most common sites of origin are the fat about the kidney, at the root of the mesentery, about the colon, and in the pelvis. They vary in size greatly, from small, well-defined nodules, 1 cm or so in diameter up to huge tumor masses. The tumors are usually lobulated, soft or semifluctuant, and of normal fat color, surrounded by a delicate connective tissue capsule. On cut sections, they are often solid and of a yellow color, but there may be areas of hemorrhage, degeneration, or sarcomatous change. Histologically, these tumors are almost wholly of normal appearing fat cells. However, a careful search should be made for malignant changes as a large percentage of reported tumors has shown such tendencies.

CONCLUSION

A case of excision of a perirenal lipoma is reported, the patient making a satisfactory recovery.

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Editorial

When to Operate in Gall-Bladder Disease

AN INCREASED knowledge of the sequence of pathologic events in acute and chronic cholecystitis makes possible certain definite statements about the optimum time for operation. In acute cholecystitis with stones a common course is the following: A stone becomes impacted in the ampulla at the cystic duct, obstruction of the submucous veins and lymphatics causes edema and swelling of the mucosa and surrounding structures. Infection supervenes later with interference with the cystic artery, veins, and lymphatics. The production of mucus causes increased pressure within the gall bladder and this, combined with the circulatory disturbance, results in local or general gangrene of the mucosa, thickening of the gall-bladder wall, and petechial hemorrhages into it. Local gangrene precedes perforation, peritonitis, or pericholecystic abscess. A thrombosis of the cystic artery may cause general gangrene of the gall bladder. In exceptional cases this occurs very early.[1-4]

The clinical course of these cases in the first few days is not that of an active infection; cultures from the contents of the gall bladder and its wall are frequently sterile and removal of the gall bladder is followed by a prompt recovery without complications. Thoughtful consideration of the pathology as described above would logically lead to a prompt operation as soon as a definite diagnosis can be made.

A second type of acute cholecystitis due to embolic infection of the gall-bladder wall with microorganisms might cause more disagreement. In the acute fulminating cases most surgeons would consider operation imperative, but many would feel that delay was indicated in the less severe infections, especially in cases seen late. There are mortality statistics available to guide us in our decision. In a collected series of 167 cases of acute cholecystitis with operation within forty-eight hours of the onset of symptoms, there was a mortality of 3.6 per cent.[5] In one series of 100 consecutive cholecystectomies for acute cholecystitis, operation within the first five days on seventy-eight cases gave a death rate of 5 per cent.[5] The deaths in this series came from suppuration and perforation in three out of the four cases. Obviously those patients who died from early operation were the fulminating cases. After five days the death rate rises rapidly in many of the published series to about 20 per cent, so this still remains a "no man's land" for further experiment and debate. Complicating diseases may compel delay at

times. We have not taken into consideration the well-known difficulty or impossibility of accurately determining the type of pathology present before operation.

In chronic cholecystitis and cholelithiasis the need for haste in saving hours or a few days is secondary, but this is a progressive disease and procrastination over a period of years has wrought deadly havoc in the past. Stones in the common duct, pancreatitis, and liver destruction are a few of the end results. Goldish and Gillespie[6] have shown that the average age of those who died following gall-bladder operations was 10 years older than those who lived. Glenn[7] states that in the New York Hospital group "in 69 patients with symptoms for less than one year no post-operative death occurred and there were 53 cases of acute cholecystitis whose symptoms had endured less than five years who survived operation. With a history of more than five years of gall-bladder disease the mortality after operation was 15 per cent."

Inflammation of the gall bladder associated with chronic jaundice has been treated in the past by most surgeons with an elastic and prolonged delay, inspired by the hope that a remission would occur during which a safer operation could be performed. Too often the jaundice has increased with more extensive liver damage and increased risk. The time has now come when these deeply jaundiced patients can be operated upon with a low mortality and freedom from postoperative hemorrhage after a few days' preparation. This change has been brought about by the use of vitamin K,[8] bile salts, transfusions, and glucose solutions.

With all these facts in mind, who can doubt that it can be truly said of gall-bladder surgeons: "The bravest are the tenderest; the loving are the daring"?

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Recent Advances in Surgery

Conducted by Alfred Blalock, M.D.

RECENT ADVANCES IN SURGERY OF THE ESOPHAGUS

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(Continued from the November issue)

HEMORRHAGE FROM ESOPHAGEAL VARICES

The development of an adequate technique for the demonstration of esophageal varices by the air-contrast relief method (Wolf, 1928; Schatzki, 1931; Brdiczka and Tschakert, 1932; Baumeister, 1933; Wachner, 1934; Holmes and Schatzki, 1935; Monauni, 1937; Plotz and Reich, 1938) has progressed side-by-side with attempts to control hemorrhage from them by rational operative means.

The largest and most extensive varices are seen in cases of thrombosis of the splenic and portal veins (Brdiczka and Tschakert; Baumeister). Lesser degrees of involvement are observed in cirrhosis of the liver, Banti's syndrome, the varices of old age, and in the congenital cases (e.g., Nochimowski, 1932). In all unexplained cases of gastrointestinal bleeding roentgenologic examination of the esophagus should be made. Wachner (1934) stated that bleeding and death may occur from varices in cases in which it is impossible to demonstrate them by roentgenography, but Holmes and Schatzki implied that by careful observation even the small varices may be detected. At fluoroscopy, differentiation must be made between varices and air pockets, esophagitis and carcinoma. Air pockets can be displaced and are readily recognized after a little experience. Varices and the walls of the esophagus about them change shape with variations in intrathoracic pressure (tests of Müller and Valsalva), but inflammatory and carcinomatous infiltrations show localized or generalized rigidity.

In a patient with Banti's syndrome Walters, Rowntree, and McIndoe (1929) attempted to control bleeding from esophageal varices by ligating the coronary vein and its branches. This was unsuccessful, but after the spleen had been removed (Rowntree, Walters, and McIndoe, 1929), the hemorrhages stopped, at least temporarily. Kirklin and Moersch (1931) reported the case of a male, aged 26 years, in whom a large spleen was removed because of bleeding from varices which were demonstrated by roentgenography; the coronary veins were not ligated because they were not distended at the time of operation. Three weeks later, the roentgenologic appearance was unchanged. These experiences led Kegaries (1933, 1934) to study the venous plexus of the esophagus in detail, following which he advised, for patients with portal obstruc-

tion who had bled from esophageal varices, the ligation of the coronary veins and the vasa breva of the spleen together with a Talma-Morrison omentopexy, with or without splenectomy, according to the ability of the patient to withstand a more extensive procedure. McClure (1934) ligated the coronary veins and, five and one-half months later, removed the spleen, with apparent relief in a case of hematemesis in Banti's disease. In a boy, aged 7 years, with Banti's syndrome, who had bled seriously from large varices which were demonstrated by roentgenograms, Rhoads and Stein (1938) ligated the coronary vessels and removed the spleen, with clinical improvement and reduction in the size of the varices as seen by roentgenography. They thought that ligation of the coronary artery might have decreased the load on the portal system, but, in view of the rich collateral distribution of blood to the stomach, this seems rather unlikely.

Kemp (1938), in a most interesting critical analysis, concluded that the crux of the situation lies, not in the ligaturing of the coronary veins and in splenectomy or tying of the splenic pedicle alone, but in the development of methods by which the dilated veins may be attacked directly. He pointed out that in cases of varix of the internal saphenous system a cure is not attained by distal ligation and that analogously distal ligation scarcely can be expected to provide much relief in cases of established varices of the esophagus, in which the columns of blood are in direct connection with the superior vena cava and unprotected by valves. Seeking to attack the problem directly, Walters (1933) suggested the injection of sclerosing solutions into or about the dilated veins through an esophagoscope. Kemp implied that it might be possible by a transthoracic approach to ligate the dilated veins proximally (i.e., the azygos and hemiazygos). If this should be done, it would be advisable, of course, as a preliminary step to ligate the coronary veins and vasa breva and to carry out omentopexy, with or without splenectomy.

In emergencies Westphal (1930) succeeded in stopping hemorrhage from varices in two cases of cirrhosis of the liver by introducing a Gottstein bag and filling it with 80 c.c. of water. The pressure was maintained for twenty-four to twenty-nine hours. He suggested the use of sedatives if the bag caused discomfort.

NEW ESOPHAGOSCOPIC INSTRUMENTS

Several instruments have been described during the past five years which may perhaps possess advantages over the older models. Korbsch (1934, 1938), Israel (1935), and Biedermann (1938) each designed instruments which make use of lenses to magnify and proximate the image. The flexible model of Korbsch is only 6 mm. in diameter and can be used to examine minutely the mucous membrane of the esophagus, the cardia, and the pyloric portion of the stomach. The telemagnifier of Israel can be focused, does not obstruct breathing, and does not in-

terfere with the passage of instruments. Biedermann's esophagoscope resembles the instrument of Jackson, but it is fitted with an optical light staff, 3.6 mm. in diameter, which is inserted through the main lumen to enlarge the field by 2.5 times and bring the image closer to the eye. He has been able to use instruments with great accuracy through the part of the tube not filled by the light staff.

Tucker (1934) described a small, long esophagoscope to be passed through and beyond a 9 mm. tube of usual Jackson design. This enabled him to enter and pass quite small zones of stenosis in the lower esophagus under conditions of improved visibility and control. In 1936 he also recommended rigid esophagogastroscopes, 6 mm. in diameter and 53 cm. long for children, and 8 mm. in diameter and 58 cm. long for adults, each provided with a combined thumb valve suction release and stopcock and a stopcock insufflation tube. He advised the examination of the esophagus with a rigid tube before the introduction of a flexible gastroscope, even if the x-ray studies had been negative, in order to rule out contraindications to the blind passage of the instrument.

Matis (1935) made use of a tube which combined the advantages of proximal (Brünings, 1909) and distal (Jackson) illumination. He felt that this helped prevent distortion and assisted him in the delicate use of instruments. In 1937, he reported that he had been experimenting with various models of flat-oval, oval, and quadrilateral distensible esophagoscopes, made in two parts which could be separated gradually after introduction. The flat-oval type seemed the best and presented many advantages over the older round tubes with lumens of fixed size.

ROENTGENOGRAPHY

Taken as a whole, the development of the art of roentgenography of the pharynx, esophagus, cardiac portion of the stomach, and the small intestine has not kept pace with the perfection obtained in examination of the lower portion of the stomach, the proximal duodenum, and the colon. However, a few roentgenologists have become interested in the special problems presented by alterations in physiology and morphology in these regions and have developed techniques which enable a careful observer to attain a high degree of accuracy in diagnosis.

Particular attention has been paid to the upper esophagus by roentgenologists who are connected with the large laryngologic centers (e.g., Pancoast, 1924, 1933; Eiselsberg and Sgalitzer, 1928; Macmillan, 1939). By a close study of the normal and abnormal appearances in the anteroposterior, oblique, and lateral positions, with and without the aid of contrast mixtures, the services of the roentgenologist have been much improved. For the mid- and lower esophagus and the region of the cardia, special attention has been directed to the importance of examining patients in several positions. The advantages of the Trendelen-

burg (pelvis-high) posture were emphasized by Palugyay (1920, 1923) and Schatzki (1931). Manges (1926) advocated the right oblique prone position for adults and the supine posture for children (Manges and Clerf, 1935). It seems likely that routine fluoroscopy in these positions will unearth the causes of many obscure thoracic and abdominal complaints. Details cannot be entered into here, but the object of the special positions is to fill the esophagus with barium and obtain good fluoroscopic and spot film views (Kruchen, 1929; Schatzki) of all portions, without too much overlapping by other structures, such as the spine, heart, and fundus of the stomach. By proper techniques, filling can often be obtained below as well as above partially obstructing lesions (Moore, 1930). Wright and Freeman (1934) obtained good filling of the unobstructed esophagus in the upright position; they directed the patient to swallow two or three mouthfuls of barium mixture during forced expiration and exposed a film immediately. In a few patients with incipient or established achalasia of the cardia better films were obtained during forced inspiration.

The gelatin or barium-filled capsules introduced for esophageal work by Hickey (1929) were found misleading by Barnes (1937), but the opaque gelatin bougies appeared to be useful. Various thick and thin mixtures of barium, many of them containing sticky substances, such as mucilage, acacia, or tragacanth, or small dry bodies like cracker crumbs have been used to assist visualization (e.g., Vinson, 1925; van Gilse, 1928; Collins, 1934; Barnes, 1937).

Macmillan (1935) was able to demonstrate all of his foreign bodies lodged in the esophagus: "If the foreign body is not located on the lateral view, a search must be made for it with the aid of a heavy barium paste, for if present the barium will adhere to it and will not be washed off by subsequent swallowing of water. If there is edema and scarification of the mucous membrane from trauma in the passage of the foreign body, there will be delay in the transit of the barium and a variation from the normal mucosal pattern, but one or two swallows of water will wash off the barium from any such area of abrasion." This method was mentioned favorably by Nissen (1934). Roentgen technique in cases of suspected perforation of the esophagus by foreign bodies or instruments was ably described and illustrated by Schiffer (1935).

Schatzki (1931) published beautiful films in various normal and pathologic conditions made by the mucosal relief (air contrast) method. This technique is particularly useful for the demonstration of esophageal varices, but Schatzki also detected the swollen folds of esophagitis, the converging folds and the small niche of an ulcer, and the slight irregularities of early carcinoma, which might not be evident by methods which depend on complete filling. The procedure was found valuable by Skinner (1937).

Moore (1930) noted that in the examination of the lower esophagus and region of the cardia the roentgenologist is chiefly concerned with the differentiation between carcinoma, cicatricial stricture, achalasia, diverticula, nonmalignant new growths, ulcers, and diaphragmatic hernias. The limitations of roentgenography, as well as esophagoscopy, in the detection of lesions of the esophagus were emphasized by Emery (1934), who stated in substance that: "Symptoms typical for carcinoma are more dependable than a negative roentgenologic and esophagoscopic report. All patients who complain of indefinite esophageal symptoms should be studied by the x-rays and esophagoscope and if symptoms persist, examinations should be repeated at least once a month until a diagnosis is made." Pesek (1934) and Barnes (1937) also advised re-examinations. The danger of misinterpreting, as achalasia, certain cases of carcinoma was pointed out by Vinson (1924). Collins (1934) noted that scarring with superimposed spasm may give an appearance almost identical to that of carcinoma. Jackson (1935) found that hiatal hernia complicated by ulcer may easily be mistaken for cancer of the lower esophagus. Many authors have emphasized that negative findings by x-ray are unreliable unless checked by esophagoscopy (e.g., Hill, 1925; King, 1936). On careful examination by Schatzki's (1931) method in his cases of ulcer, Macmillan (1935) observed under the fluoroscope considerable spasm of the esophagus and often a fleck of barium remained in the affected area. However, many esophageal ulcers do not show the deformity and niche which are so typical of duodenal ulcer. If epigastric or substernal pain persists, esophagoscopy is always indicated (Jackson, 1929).

The possibility of obtaining information as to the state of adjacent organs or tissues by roentgenographic studies of the barium-filled esophagus has been recognized for many years. Recent contributions, particularly in relation to deviations caused by an enlarged left auricle in mitral and aortic disease, have been made by Rösler and Weiss (1925), Brown and McCarthy (1935), Schwedel (1937), and Conrads (1937). Graham, Singer, and Ballon (1935) illustrate a diminution of the deviation of the esophagus as a result of a Brauer operation in a case of large rheumatic heart. Deviations, stenoses, and fistulas due to various extrinsic changes, such as pulmonary tumors, neoplasms of the vertebrae, cold abscesses, aneurysms of the great vessels, a high right-sided anomalous position of the aorta, and pleural exudates, have been noted. Fell and Goldschlag (1935) and Doig (1936) report cases in which enlarged or fibrosing tuberculous mediastinal lymph nodes displaced the esophagus. In one of Doig's cases the angulation was so abrupt as to cause dysphagia. He suggested the possibility of a thoracoplasty for its relief, should the dysphagia become serious. Blalock (1935) noted that, at least in dogs, total pneumonectomy, without associated paralysis of the diaphragm, was followed by a marked deviation of the esophagus to the side from which the lung was removed.

The occurrence of peristalsis in the esophagus has been demonstrated repeatedly by both physiologists and roentgenologists, but the question of antiperistalsis (during vomiting or as the result of obstruction or spasm) is still subject for debate. Largely on the basis of roentgenologic observations in human beings and in dogs, Becchini (1935, 1937) and Aprile (1937) agreed with several earlier authors that true antiperistalsis occurs not infrequently. Dahm (1938), on the other hand, using the roentgenkymograph, demonstrated to his own satisfaction that Schwartz (1924) was correct when he interpreted the apparent antiperistalsis as a reflux of barium, which, encountering obstruction, passed backward through the lumen as the advancing forward wave of peristalsis approached and passed the obstructed region.

Jackson and Jackson (1933) pointed out that lesions of the esophagus frequently cause cough by reflex irritation or by entrance of esophageal contents into the respiratory tree. In the latter event fluoroscopy shows barium entering the bronchi through a tracheoesophageal or bronchoesophageal fistula or by overflow from an obstructed esophagus directly into the larynx and trachea. Other causes of such an overflow are bulbar paralysis, compression of the ninth and tenth nerves at the base of the skull, and myasthenia gravis.

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Book Reviews

Pye's Surgical Handicraft. Edited by Hamilton Bailey, M.D. Ed. 11, revised. Cloth. Pp. 500, with 362 illustrations. Baltimore, 1939, Williams and Wilkins Company. $6.

This small compendium of hospital procedures which has been republished periodically for fifty-four years has undoubtedly introduced many a surgeon into the intricacies of the surgical art. When the reviewer was first a surgical house officer, an earlier edition of this book was constantly by his bedside and was consulted frequently. It contains an extraordinary variety of practical materials, all dealt with concisely and all pertinent to the management of surgical cases. It is illustrated profusely. A large number of contributors writing on subjects of their special interest lends assurance that the subject matter is dealt with in an up-to-date and authoritative manner. This book should have a wider acquaintance among American readers.

Diseases of the Nose and Throat. By Charles J. Imperatori, Professor of Otolaryngology, New York Polyclinic Medical School and Hospital, and Herman J. Burman, Adjunct Professor of Otolaryngology, New York Polyclinic Medical School and Hospital. Ed. 2. Pp. 700, with 480 illustrations. Philadelphia, 1939, J. B. Lippincott Company. $7.

The first edition of this text was published in 1935. It was so well received that the authors have brought this second edition up to date to include the progress made in rhinolaryngology.

This text is an outline of the subject of nose and throat disease. The authors indicate that it represents essentially the course which has been given to matriculates of the New York Post-Graduate Medical School of Columbia University. In this outline form, symptoms, diagnosis, and treatment are considered first and etiology and pathology are placed at the end of each discussion. All of the information is condensed in a series of statements in paragraph arrangement. For example, the subject of acute rhinitis is discussed in five pages in the following order: symptoms, diagnosis, treatment, complications, pathology, etiology, prophylaxis, and treatment. There is no critical evaluation of ideas other than the authors' own. Thus it lacks the completeness of the orthodox text.

The outline is rather complete. There are fifty-two chapters in all. There is a discussion of sinusitis as a focus of infection, physical therapy and radiation, allergy, manifestations of general diseases, and laboratory aids. The illustrations are ample for a treatise of this type and are practical ones. Considerable detail is given to office procedure and the technique of treatment. The subject of pathology is proportionately considered.

For the student and practitioner this text offers a very readable and practical outline of what the majority of rhinologists and laryngologists accept as fact, carefully arranged by these two well-known specialists.

Septische Chirurgie. By Dr. Med. Habil. Fritz Schorcher, Universitat Munchen. Paper. Pp. 270, with 74 illustrations. Leipzig, 1938, Johann Ambrosius Barth. RM 20 unbound, RM 21.50 bound.

This small monograph deals with the various suppurative processes which fall within the province of the general surgeon. The subject matter has been discussed in an orthodox and authoritative manner. The drawings of surgical exposures are particularly good. One weakness of the text relates to rather sketchy management of a number of disorders, but the most serious omission is failure to include any discussion of the pertinent literature or to list any bibliographic references. The author has brought together in one text the surgical problem of infection in its various manifestations.

Surgical Applied Anatomy. By Sir Frederick Treves. Ed. 10 revised by Lambert Rogers, M.D. Fabricoid. Pp. 748, with 192 illustrations, including 66 in color. Philadelphia, 1939, Lea and Febiger. $4.50.

Applied anatomy, according to the late Sir Frederick Treves, has a twofold function. "First, it serves to give a precise basis to those incidents and procedures in practice that more especially involve anatomical knowledge; and secondly, it endues the dull items of that knowledge with meaning and interest by the aid of illustrations drawn from common medical and surgical experience."

With these objects in mind, the present reviser, Mr. Lambert Rogers, has done a creditable job. The contents are divided into six parts, covering the head and neck, the thorax, the upper extremity, abdomen and pelvis, lower extremity, and the spine and spinal cord. The book is intended mainly for the use of students preparing for their final examinations in surgery. The more practical details of anatomy are stressed. The clinical and pathologic examples are well chosen. Detailed accounts of the anatomy of hernia and surgery of arteries are left to larger systematic treatises.

The book is pocket size, but the print is very readable. The illustrations and diagrams are excellent. An index is appended. Undoubtedly this popular volume will go through many future editions.

Diseases of Women. By Ten Teachers. Edited by Sir Comyns Berkeley, M.S., M.D., B.Sc., Lond., F.R.C.S., etc., Consulting Obstetrical and Gynecological Surgeon to the Middlesex Hospital. Ed. 6. Cloth. Pp. 492, with 158 illustrations. Baltimore, 1938, Wm. Wood & Co. $6.

This new work has been produced by collective authorship, the ten contributors being prominent figures in gynecology and obstetrics, not only in England but also in this country. All in all, the work is fairly comprehensive, although several subjects are presented in a rather cursory manner, which may be sufficient for the needs of the medical student but certainly not for the gynecologist.

The subject is presented in the usual logical manner, the opening chapters being devoted to anatomy, embryology, and physiology of the generative tract. A discussion of puberty, amenorrhea, menopause, and menstrual irregularities then follows.

The sections on the physiology and disorders of menstruation have been rewritten, bringing up to date the newer concepts in the field of reproductive endocrinology.

The discussion of malpositions of the uterus includes the recommendation of the Aveling repositor for the use of uterine inversion, which to my knowledge is not the accepted procedure in this country; however, it would seem worthy of trial.

The next section deals with inflammatory processes of the generative tract, which includes a discussion of chronic metritis, chronic subinvolution, and simple hypertrophy of the uterus, conditions as a rule not sufficiently dealt with in most textbooks.

Considerable space is given to the consideration of the benign and malignant tumors of the uterus and ovary. The chapter on carcinoma of the cervix is fairly complete, although the portion devoted to pathology would seem too brief. The chapter devoted to tumors of the ovary is comprehensive, clearly presented, and fully illustrated.

The remaining chapters are mainly taken up with the consideration of extrauterine pregnancy, contraception, and operative procedures.

The book is written in a lucid, concise style, which is characteristic of so many English authors. The type is clear, and, although the illustrations are numerous, they are not particularly well done. The value of the publication would be enhanced by the addition of a bibliography. The work is quite worth while and should prove of considerable value to the student of gynecology.

Treatment by Manipulation. By A. G. Timbrell Fisher, N.C., M.B., Ch.B., F.R.C.S., England. Ed. 3. Cloth. Pp. 255, with 68 illustrations. New York City, 1939, Paul B. Hoeber, Inc. $3.75.

Timbrell Fisher stated in his preface to the third edition of this monograph that his purpose was to call to the attention of the medical profession the great importance and value of manipulative treatment in the management, in carefully selected cases, of certain sequelae of injuries and diseases, particularly affecting the joints, muscles, tendons, and fascia. This purpose has been adhered to in the third edition, which has been enlarged and altered to bring the subject matter up to date.

The chapter on osteopathy deals with the history of and fallacies in this cult. The difference between manipulative surgery, as practiced by qualified medical practitioners, and osteopathy, a revolutionary system of medicine, based upon a theoretical hypothesis unsupported by scientific evidence, is clearly drawn.

The three chapters devoted to a discussion of the pathology, the prevention, and the diagnosis of adhesions are excellent and emphasize fundamentals with which those who attempt to treat disturbances of the joints, tendons, and fascia should be thoroughly familiar. Perhaps the most important section of these chapters is that which deals with the role of early movements in sprains, fractures, arthritis, and other conditions leading to limitation of movement and deformity in joints. The views expressed are based on an extensive experience and intelligent observation and are worthy of study by every practicing physician, be he specialist or general practitioner.

The chapter dealing with the general principles of manipulative treatment and the application of these principles to individual joints presents subject matter in a clear-cut, systematic manner. The technique of the manipulations to be carried

out for each joint is carefully described and clearly demonstrated by the accompanying illustrations. The final chapter on aftertreatment is brief but full of important and useful information.

Fisher has handled a delicate subject in a masterly manner; he has been conservative in the claims he makes for treatment by manipulation and has clearly pointed out its limitations and its dangers. This book has placed treatment by manipulation in its legitimate place in the armamentarium of the practicing physician, and makes an excellent case for including instruction in this important therapeutic measure in our medical curriculum.

Surgical Pathology. By William Boyd, M.D., University of Toronto. Cloth. Pp. 886, with 476 illustrations and 15 color plates. Philadelphia, 1938, W. B. Saunders Company. $10.

The fourth edition of Boyd's *Surgical Pathology* has been revised to cover all the important new contributions in this field. The book is more than a surgical pathology. It is in reality a guide to surgical procedures which have been found to be of practical value. In many chapters there is a discussion of theories of disease processes. The presentation of the clinical features of various disorders makes the book one which has an appeal to the practicing surgeon. The author has a pleasant, clear style so that the book is easy to read. In attempting to cover detailed gross and microscopic pathology in a volume of this size, it is necessary to be dogmatic. For this reason controversial issues in the pathologic field sometimes are avoided or dismissed with little discussion. Occasional discrepancies are bound to creep in such as that on hypernephroma. On page 192 "hypernephroma is a tumor consisting of adrenal tissue," but on page 440 the term is used "with the clear understanding, however, that the use of this name does not commit one to the adrenal theory of the origin of these tumors."

There are many excellent new illustrations. It is disappointing to find some rather stereotyped drawings retained from the earlier editions. These should be replaced by photomicrographs in future editions.

The general outline of the presentation is for anatomical regions. The pathologic conditions of surgical interest in each organ are taken up in an orderly fashion. The active surgeon will find the book a valuable *vade mecum*.

The section on adrenal conditions might be revised for a clearer presentation. In view of their increasing frequency a section on the tumors of the lungs and bronchi would seem to be in order. With these minor exceptions, this edition of *Surgical Pathology* answers most of the questions of interest to the surgeon. It stands between the books on general pathology and the monumental work of Ewing on *Neoplastic Disease*.

The Rectum and Colon. By E. Parker Hayden, Assistant Surgeon and Chief of Rectal Clinic, Massachusetts General Hospital. Cloth. Pp. 434 with 169 illustrations. Philadelphia, 1939, Lea & Febiger. $5.50.

This well-printed and illustrated book apparently reflects the experience and beliefs of the Rectal Clinic of the Massachusetts General Hospital whose staff is composed entirely of surgeons who have been functioning in this capacity for varying periods of time for the past ten years. The author states in the preface that he has made an effort to give each pathologic condition that degree of emphasis which its relative importance warrants without attempting to include in the bibliography all of the important references bearing on a subject.

One is struck with the forthright exposition of the subject. No effort is made at finality of opinion, but the various conditions are described and discussed with candor and at the same time reflect the author's personal viewpoint. The reader, whether he is seeking information on the subject or simply is interested in the viewpoint of the Clinic can be assured good reading. For instance, the experience of the Massuchusetts General Hospital in ulcerative colitis is well given in the quotation from McKittrick's work. The influence of the late Daniel Jones is shown in the sound consideration of the management of cancer of the rectum.

For the general practitioner the book offers a systematic and entirely sound approach to the management of the important lesions involving the rectum. The injection treatment of hemorrhoids is discussed at length with, I believe, insufficient emphasis on the palliative rather than curative nature of the results. The descriptions of the operative methods for hemorrhoids are good but fail to give the clamp and cautery method what some might think a fair appraisal. A careful and critical discussion of oil-soluble anesthetics as well as their formulas are given. The advocacy of operation for acute thrombosis of hemorrhoids is against the usual practice but is sound. Prolapse of the rectum is well considered. In the discussion of lymphogranuloma inguinale many surgeons of experience would take real exception to the advisability of resection of the rectum for its cure. The omission of the mention of the Strauss proctoscope in the description of those instruments or of Sudeck in the discussion of the critical point of ligation of the superior hemorrhoidal artery is not important except where others are given credit for their contributions.

In general the above criticisms are not fundamental, for the principles and practice outlined in this book on the colon and rectum are sound and should be of great value to the general practitioner and surgeon who needs information on the subject.

End Results of the Treatment of Gastric Cancer. By Edward M. Livingston, M.D., and George T. Pack, M.D. Cloth. Pp. 179, with 145 illustrations, New York, N. Y., 1939, Paul B. Hoeber, Inc. $3.

This unusual and remarkable book will lend considerable encouragement to the problem of cancer of the stomach. The authors have summarized the world's literature of 14,000 reported gastric resections. The average mortality rate for resection is slightly under 20 per cent. Of patients who survive resection, 20 per cent will be alive and well after five years. To those familiar with the papers of Welch and of Friedenwald, this is encouraging information. The lamentable matter, which is not always apparent in surgical figures, but which the authors have been at pains to point out, is that the nonoperable, as well as the nonresectable, group is very large.

The plan of the book is unique and should prove a good prototype for surgical monographs. The authors have departed widely from the usual orthodox plan of writing. Tables and concise listing of pertinent information should make the book very useful, not alone to surgeons but to internists and practitioners as well. The choice of bibliographic references is good. Without question, this is one of the best monographs on cancer of the stomach that has ever been published and should be read widely.